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POTASSIUM DICHROMATE AS A WITCHDOCTOR'S REMEDY

FIVE CASES OF POISONING

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Chromic acid and its alkaline salts have many industrial uses particularly in tanning, dyeing and printing[1]. Workers in these occupations are exposed to a fine dust, or mist, of chromates which, before the days of adequate protection, produced ulcers of the nasal septum ('chrome holes') or dermatitis of the exposed skin. There is also evidence that chrome workers are more susceptible to carcinoma of the lung than the general population[2]. In contrast to their local action on skin or mucous membranes, chromates act as virulent poisons when they are absorbed into the body. Since Wilson[3] described the first case in 1844, many reports of accidental or suicidal chromate poisoning have appeared in the literature.

In this paper it will be shown that chromate poisoning is also seen in South Africa and that it occurs in an unusual manner: amongst Africans potassium dichromate is regarded as an effective remedy for many diseases. It is not only dispensed by witchdoctors throughout the country, but it is freely used by laymen as an emetic and a purgative. It is often taken in massive dosage and then rapidly causes death, but where less of the substance is absorbed it produces an illness whose true nature may escape recognition, since the patient forgets or withholds the fact of having taken the poison.

CASE REPORTS

Case 1: G.M., an African male aged 20 years, had consulted a European doctor about a week before admission for a bout of vomiting. He returned 4 days later, this time jaundiced, and he was then admitted to hospital.

Examination revealed a very ill young man. He was stuporose, answered questions in a confused manner and grimaced when examined. He hiccoughed continuously. He was deeply jaundiced, his sclerotics being a deep golden colour. The abdomen was distended; the lower edge of the liver could not be felt and its upper border was not displaced. The tongue and skin were dry. Temperature 99°F, pulse rate 100 per min., blood pressure 140/60 mm. Hg. There were no burns in the mouth or throat. The urine contained 3 red blood corpuscles per high-power field, with bilirubin and an excess of urobilin present; specific gravity 1010.

On the 2nd day the patient's breathing became fast and hissing (Kussmaul in type), and a fine urea frost appeared on his brow. For the next 7 days there was little change in his clinical state. The temperature, jaundice, mental clouding, hiccough, hissing breathing, urea frost and tympanites persisted; from time to time he shivered violently. On the 4th day he passed a pasty stool; on the 5th day he developed oedema of the feet and an erythematous rash appeared on his arms and thighs.

On his 8th day in hospital he showed clinical improvement; the mental clouding lifted, he hiccoughed less frequently, his breathing became normal and his jaundice less evident. By the 16th day he was quite well; his skin was desquamating in the distribution of the rash.

Laboratory Studies. Traces of chromium were found in the urine and gastric contents on admission, but none could be detected 3 weeks later. The urinary output was 0·5 l. on the first day and 0·5—1·3 l. daily for the next 10 days. At the 30th and 40th day the specific gravity after 18 hours of fluid restriction was 1023 and 1027 respectively. On admission the blood-urea level was 348 mg. % (Table I) and this fell to normal limits in 2 weeks; the CO_2-combining power of the blood ranged between 39 and 29 c.c. for the first 14 days and became normal 4 weeks after admission. During the first week blood-potassium levels were low but other electrolytes were not disturbed. The level of alkaline phosphatase was high on admission; the liver-function tests though abnormal did not change significantly with clinical recovery. Aspiration biopsy of the liver performed on the 14th day revealed moderate haemosiderosis but no other significant pathological change. Malaria, yellow fever and Weil's disease were excluded by appropriate blood tests.

Treatment For the first week what little nourishment the patient took by mouth consisted of milk and a solution of 1/6 molar lactate. He was maintained on intravenous fluids, 5% dextrose-water, 5% dextrose-saline and 1/6 molar lactate, the quantity restricted to 1 l. in excess of the daily urinary output. In addition he received vitamin K, aureomycin, choline, methionine and BAL.

Summary A young man presenting with the picture of hepato-renal failure was found to have traces of chromium in the urine and gastric contents. He recovered and was apparently left with no permanent renal or hepatic damage. The patient and his relatives steadfastly denied that he had taken potassium dichromate or any Native medicine.

Case 2: J.T., an African male aged 33 years, had received *ndonya* (potassium dichromate crystals) from a witchdoctor 6 days

previously and shortly after drinking a solution of the crystals had vomited violently. He had passed no urine for 48 hours before admission.

On examination he showed similar clinical features to case 1, namely hiccough, Kussmaul breathing, and abdominal distention. He differed only in being less jaundiced and mentally confused. His bladder contained about 150 c.c. of urine. Chromium was detected in both gastric contents and urine. He was uraemic and acidotic, but his blood-potassium level—unlike that of case 1—lay within normal limits (Table I). He deteriorated rapidly. Within 3 days he became drowsy and more deeply jaundiced; he remained persistently anuric, urea frost appeared about his face and he died on the 4th day after admission. As in case 1, there were no burns about the mouth or throat.

From the 1st day he was fed with 1 l. of Bull's emulsion[4] through an intragastric drip and was treated with vitamin K, aureomycin, choline, methionine and BAL. On the third day, ACTH, 20 units 6-hourly, was administered by intravenous drip and this was continued until his death.

Post mortem Examination (*Dr. V. Kemp*). The positive macroscopic findings consisted of a few petechial haemorrhages in the stomach, bladder and renal pelves. There were no burns in the mouth or oesophagus and there was no erosion of the gastric mucosa.

Histological Examination (*Dr. B. J. P. Becker*). (1) Sections of the kidney showed the presence of acute tubular necrosis affecting both upper and lower nephrons. The lumen of the tubules was occupied by intensely-staining eosinophilic granular casts. (2) Sections of the liver showed the presence of focal necrosis with many bile thrombi and bile casts; the necrosis was massive in places. Non-necrotic portions of the liver lobule showed feathery degeneration and focal fatty change. (3) Sections of the lung showed no significant pathological change.

Chemical Analysis (*Dr. B. W. Marloth*):

Organ and weight	*Chromium calculated as potassium dichromate*
Liver, 1,050 g.	0·90 gr.
Kidneys, 320 g.	0·23 gr.
Stomach and contents, 300 g.	0·14 gr.
Intestines (large and small), 2,086 g. ..	0·47 gr.
Total	1·74 gr.

Case 3. M.M., an African male aged 50 years, had been given some medicine by a witchdoctor 5 days before admission which he used as an enema. Shortly afterwards he started purging; within 1 or 2 days his stools became bloody and he began to vomit. On examination he showed the now-familiar signs of jaundice, urea frost, drowsiness, 'acidotic' breathing, hiccough and abdominal distention; on the 5th day in hospital he developed a maculopapular rash over his arms and legs. In addition he showed certain features not previously encountered: at the height of his illness he bled from the gums and mouth, developed a brisk haematuria and his haemoglobin level fell from 12·1 g % on admission to 7·0 g. % within a week (Table I). His urinary output was never less than 0·5 l. daily and he recovered in 3 weeks.

In this case the excreta were not analysed and the 'medicine' was not available but the patient was certain that it had contained *ndonya*.

Case 4. W.T., an African male aged 30 years, had taken a black pill given him by a friend to cure a stomachache one week before admission. He complained of vague pains and that his 'eyes were yellow'. He resembled the previous cases in being jaundiced and confused but was never uraemic or acidotic. His urinary output remained over 1 l. per day and his urine contained bilirubin but no cells or casts. Though chromium was detected in the urine he could not remember experiencing an episode of vomiting or purging. He was treated like case 1 and although his general state improved he remained jaundiced for 3 months and was almost certainly developing cirrhosis of the liver when he was discharged.

Case 5. H.S., an African male aged 45 years, had drunk a solution containing ½ teaspoonful of crystals given him by a witchdoctor 24 hours before admission; 5 minutes later he vomited 4 times, and during the night vomiting recurred and he collapsed. He had not passed urine during the 24 hours since taking the medicine. Apart from drowsiness no abnormality was found on physical examanation and there were no burns of the mouth or throat. His bliadder contained about 60 c.c. of urine which showed 20 red blood corpuscles and a few granular casts per high-power field; blood urea was 90 mg. %. The crystals produced by his wife were identified on analysis as potassium dichromate.

He was treated by Bull's method and started passing urine the following day. Within a week he was well and his blood urea had returned to normal limits (Table I).

DISCUSSION

Several post-mortem examinations on cases of chromate poisoning are recorded in the literature[5, 6, 7]. The microscopic findings are not striking. They consist of petechiae and superficial erosions of the stomach, and ochre discolouration of the liver and kidneys due to chromic oxide. Where the poison has been used as an enema, similar superficial or deeper lesions are found in the rectum[8]. Macroscopically, the chief lesions are found in the kidneys, which show all degrees of damage from degeneration to necrosis, and in the liver, which usually shows degeneration. The clinical and pathological findings in the 5 cases reported here confirm that potassium dichromate may produce degrees of renal and hepatic damage ranging from the reversible to actual death of tissue. Moreover the action of the poison does not appear to be corrosive in the mouth, stomach or rectum.

The clinical features of poisoning vary with the amount of chromium absorbed and the following description is

TABLE I. LABORATORY INVESTIGATIONS IN 5 CASES OF POTASSIUM DICHROMATE POISONING

Case Number	*Hospital Day*	*Blood Urea (mg.%)*	*Serum Bilirubin (mg.%)*	*CO_2 Combining Power (c.c.)*	*LFT*			*AP*	*Blood Sugar (mg.%)*	*PI%*	*Serum Na (mg.%)*	*Serum K (mg.%)*	*Hb (g.%)*	*WBC*	*ESR*
					TT	*TF*	*TA*								
1	2	348	16	39	1·5	—	+++		105	85	335	14·8	16·2	9,300	
	6	119		29	1·0	—	+++		105	94	335	14·4			
	28	16	9	38						100	357	22·0			
	36		2·7	49	1·0	—	++				336	18·8			
2	1	428	3·6	29							300	25·0	17·0	11,400	7
3	1	470	24										12·1	10,200	
	7	120	6										7·0	13,700	
4	7	21	16·2					21·5	110	55	305		15·6	11,900	33
	90	39	18·0					22·2		95					4
5	1	134													
	3	66													
	6	20													

LFT = liver function tests. TT = thymol turbidity (units). TF = thymol flocculation. TA = Ucko's modification of Takata-Ara reaction. AP = alkaline phosphatase (units %). PI = prothrombin index. Hb = haemoglobin. WBC = white blood cells (per c.mm.). ESR = erythrocyte sedimentation rate.

based on case records in the literature and the experience of these 5 cases:

A large amount of poison rapidly produces uncontrollable vomiting and diarrhoea which proves fatal within a few minutes or hours; these cases of acute poisoning are usually first seen by pathologists.

The patients more likely to be encountered by clinicians are those who have absorbed less poison and have recovered from the episode of vomiting and purging. The mildest cases develop transient oliguria and azotaemia, due to slight renal damage or a state of extra-renal uraemia (case 5 in this study.) Those who have sustained more serious hepatic or renal damage develop jaundice or hepato-renal failure after the lapse of 5—7 days. In these delayed, or subacute, cases the bout of vomiting and diarrhoea is apparently not always severe, as in case 4 in the present series.

The diagnosis rests on a history of taking the poison, the findings described, and identification of chromium in gastric contents, faeces or urine. In cases 1 and 2 chromium was still detectable in the urine and gastric contents 5—7 days after taking the poison and in spite of considerable vomiting. The history of taking potassium dichromate may not be forthcoming in Africans for 2 reasons: firstly, the patient or his relatives may be covering some person unqualified to administer the drug and, secondly, the chromate may have been so disguised by the addition of herbs that the patient is ignorant of the contents of the witchdoctor's 'medicine'.

The differential diagnosis in cases of subacute poisoning is that of jaundice, or jaundice in association with uraemia (hepato-renal failure). Haemolytic jaundice can be excluded by a blood count (anaemia, reticulocytosis) and the presence of bilirubinuria; obstructive jaundice due to gall-stones or neoplasm is not commonly seen in this hospital. It is therefore reasonable to consider poisoning as a probability in every case of jaundice in Africans, particularly when it is associated with marked azotaemia. In the past 3 years several cases of hepato-renal failure have been seen in this hospital for which no adequate explanation has been found clinically or at post-mortem examination. Since liver disease is common amongst Africans[9] it is possible that they develop hepato-renal failure more commonly than white races; in such cases, however, the possibility of poisoning cannot be definitely excluded since potassium dichromate may produce so few characteristic lesions at autopsy.

Judging by the outcome in these 5 cases, the immediate prognosis seems to depend on the urinary output. In case 2 the patient was persistently anuric and suffered such extensive renal and hepatic damage that it is doubtful whether he would have benefited by no-matter-what therapy. The patients who recovered all had reasonably good urinary outputs. That chromium may produce chronic or progressive parenchymatous changes is borne out by Major's case[6], in which death eventually took place from uraemia, and case 4 in this series, in which jaundice was still present 3 months after the poison was taken. As corrosion is not a prominent post-mortem feature and since chromium is found in the stomach for several days after poisoning, gastric lavage seems to be indicated when the poison has been taken by mouth.

The following information about potassium dichromate has been gathered from witchdoctors, African patients and vendors of African medicines. Known as *ndonya* potassium dichromate is an old-established African remedy. How it gained a place in the armamentarium of witchdoctors, who use mainly herbal remedies, is not known; presumably it was introduced by European chemists at one time. Certainly it is a powerful emetic and purgative and this, together with the attractive orange colour of the crystals, may be the reason for its popularity amongst the Africans.

Witchdoctors consider it an effective remedy for many of the diseases which they diagnose. It is mixed with herbs and crushed into a grey powder which is taken by mouth or as an enema; sometimes it is mixed with copper sulphate and heated over a fire; those who cannot afford to consult a witchdoctor prepare a solution of the crushed crystals in warm water which they drink or use as an enema. Witchdoctors apparently are aware of its poisonous properties and use it cautiously, but the method and dosage employed in preparing their medicines is so haphazard that it seems to be a matter of chance whether a patient will receive a lethal dose or not.

The frequency of chromate poisoning in this country can be roughly assessed from various sources. At the Government Chemical Laboratories, Johannesburg[10], a survey was made of all deaths from poisoning in South Africa from 1948 to 1952. It was found that, after arsenic, chromium was the commonest metallic poison, that it caused 21 deaths in that period, and that chromate poisoning was increasing in frequency. In 1953 the Johannesburg inquest magistrate dealt with 15 inquests on deaths from chromate poisoning[11]. The 5 cases reported in this paper were encountered over a period of 3 years but the last 2 were seen in the space of one month and doubtless many cases of 'jaundice' have escaped true diagnosis.

Potassium dichromate is widely used by Africans in this country; of the 5 patients reported here one came from Natal and another from the Northern Transvaal. So great is the demand for it that one wholesale manufacturer of African remedies buys the drug by the hundredweight. It is available to the public, as are also copper sulphate, cantharides and mercury, at Native chemists' and at shops with a predominantly African clientele.

CONCLUSION

Apart from its action as an emetic and a purgative, potassium dichromate has no therapeutic value. There can be no justification for allowing the sale of so dangerous a drug when many safe purgatives are available on the market. Its use by witchdoctors has been condemned from the bench[11] and it seems that the time has come for official action to protect ignorant Africans from this and similar 'remedies'.

SUMMARY

1. In South Africa potassium dichromate is used as a medicine by African witchdoctors and laymen and commonly produces poisoning.

2. The clinical and laboratory features of subacute poisoning are described in 5 patients, including results of a liver biopsy in one case and post-mortem findings in another.

3. The differential diagnosis is discussed. It is suggested that in all cases of hepato-cellular jaundice in Africans potassium dichromate poisoning should be considered.

4. The high incidence of chromate poisoning in South Africa is indicated and it is suggested that the sale of this and similar 'remedies' be restricted.

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DUODENAL STENOSIS DUE TO PYONEPHROSIS

A CASE REPORT

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In the September volume of the *British Journal of Urology* a case is reported in which a fistula developed between the kidney and the duodenum as the result of a stag-horn calculus and perirenal sepsis. The author points out that only 8 other such cases have been reported in the literature, but the following type of complication is even rarer and therefore probably deserves recording.

A married European female of 67 complained of abdominal pain, flatulence and pain through to her back on the right side, typical of gall-stone dyspepsia. Cholecystograms confirmed the diagnosis of cholelithiasis and cholecystectomy was performed. Her gastric symptoms improved markedly, but she was left with a pain in her right loin and right lower chest region, and attacks of nausea whenever there was exacerbation of symptoms.

It was only at this stage that I started searching for a further cause, and on further detailed questioning she disclosed the fact that she also had dysuria and frequency during attacks, and rigors on a few occasions.

The urine contained numerous pus cells, red blood-cells and many motile bacilli.

An I.V.P. showed no concentration of dye on the right side, but a normal left kidney, and a retrograde pyelogram on the right side showed gross destruction of renal substance.

Nephrectomy was exceedingly difficult in a very short and fat elderly lady, even after removal of the 12th rib. The kidney was about two thirds the normal size and grossly adherent to the peritoneum, inferior vena cava and duodenum, and in fact I considered myself extremely lucky in getting away without a duodenal fistula developing.

On the day after the operation I showed the specimen at a medical meeting and no one could recognize it as kidney. The pathology report was pyonephrosis with areas of spread of the infection into the renal tissues right through to the surface of the kidney.

Convalescence was smooth, and the patient was very well for 6 months, when she started getting pain of a colicky nature after food, sometimes followed by vomiting. This became worse and eventually Dr. J. A. Louw of Libode referred her back to me with a clinical diagnosis of pyloric obstruction, probably due to carcinoma. A barium meal showed a dilated stomach, no sign of carcinoma, and incomplete obstruction in the second part of the duodenum.

After dieting and stomach washes, she improved so much that she refused operation, but was back a week later again with obstruction.

At operation I found scarring round the second part of the duodenum, and no sign of a tumour.

I performed a simple posterior gastro-enterostomy and now, almost 6 years later, she is still quite fit and enjoys her meals.

SUMMARY

A case is related in which gall-bladder pathology masked the presence of a kidney severely damaged by pyonephrosis.

Six months after nephrectomy the duodenum stenosed up as a result of extensive perirenal infection, requiring gastro-enterostomy.

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VAN DIE REDAKSIE

BEREIDING VAN GENEESMIDDELS EN DIE N.H.S.

In sy toespraak as voorsitter van die Britse Farmaseutiese Kongres te Oxford het H. Davis 'n interessante verslag gelewer en sy mening uitgespreek oor die geneesmiddelebereiding van die Britse *National Health Service*.

Die koste van geneesmiddels word nou grotendeels deur die staat gedra wat derhalwe regstreeks belang daarin stel. As gevolg van die N.H.S. is dit moontlik om statistiek, oor die verskillende soorte geneesmiddels ensovoorts, te versamel. 'n Span ondersoekers in diens van die Britse Departement van Gesondheid is met hierdie taak belas. Voorskrifte vir middels wat van twyfelagtige waarde of oneties van aard is word ondersoek, asook buitensporig duur handelsmerke van standaard middels. Sekere beginsels is neergelê wat nagekom moet word i.v.m. die bestelling van preparate; sommige preparate mag sonder voorbehoud voorgeskryf word maar preskripsies vir middels wat onder seker groepe sorteer word beperk en ontmoedig. Onder laasgenoemde middels is 'n swetterjoel preparate—almal dieselfde—met name wat vir beide geneesheer en farmaseut hoofbrekens gee en die apteker dikwels in geldelike verleentheid stel.

Aangesien die voorskrifte vir bykans 90% van die bevolking deur die kantore hanteer word, wat die pryse beheer, is voldoende statistiek beskikbaar om die terapeutiese rigtings aan te dui. Davis gee in tabelvorm die persentasies en syfers aan wat verkry is deur ontleding van duisende voorskrifte. Soos verwag kon word is bevind dat antibiotika, sulfonamides, barbiturate en uitwendige middels op besonder groot skaal voorgeskryf word; lakseermiddels, purgeermiddels en teensure word gekenmerk deur die groot verskeidenheid handelspreparate wat bestel word. Betekenisvol is die daling in mengsels wat aangemaak word en die styging in pille wat voorgeskryf word. Dit weerspieël die verwisseling van poliifarmaseutiese preskripsies wat die plaaslike apteker aanmaak met klaar-bereide preparate wat die handelsfarmaseut bemark.

Soos dit steeds moeiliker word vir diegene wat preskripsies uitskrywe om tred te hou met die geweldige styging in die getal nuwe terapeutiese preparate en hul veelsoortige name, word die apteker deur sy mediese kollegas oor baie aspekte van hedendaagse geneeskunde geraadpleeg. Met die snelle uitbreiding i.v.m. die vervaardiging van nuwe middels deur die farmaseutiese bedryf, met wie die Britse Departement van Gesondheid in noue voeling en samewerking is, moet die apteker op hoogte van sake

EDITORIAL

PHARMACY AND THE N.H.S.

In his address to the British Pharmaceutical Conference at Oxford the chairman, H. Davis, gave an interesting report and his personal opinion on the pharmacy of the British National Health Service.[1]

The cost of medicines today is largely borne by the State, which therefore has a direct interest in the matter. Under the N.H.S. it is now possible to obtain particulars of the types of pharmaceutical products supplied and other statistics concerning medicine prescribed. An investigational unit of the British Ministry of Health exists for this purpose. Investigations have been made into the prescribing of drugs and medicines of doubtful value or of unethical character, and unnecessarily expensive brands of standard drugs. Certain principles have been formulated on which various preparations may be ordered; some can be freely prescribed, but the prescribing of drugs in certain categories is restricted and discouraged. Many of the latter represent a multiplicity of similar preparations with names burdensome to practitioner and pharmacist alike, and often a source of financial embarrassment to the chemist.

As the prescriptions for the treatment of over 90% of the population pass through the pricing offices large samples can be studied to get a picture of therapeutic trends. Davis presents in tabular form actual percentages and absolute figures obtained on analysis of thousands of prescriptions. It is not surprising that antibiotics, sulphonamides, barbiturates, tonics and external applications are found to be very extensively prescribed; laxatives, purgatives and antacids are characterized by the large variety of proprietary preparations that are ordered. There has been a significant decline in the proportion of mixtures dispensed and a marked increase in prescrthe ibing of tablets. This reflects the change-over from polypharmaceutic prescriptions made up by the local pharmacist to the ready-made preparations of the manufacturing chemist.

As it becomes increasingly difficult for the prescriber to keep pace with the ever-growing number of new therapeutic preparations and their multifarious names the pharmacist is consulted by medical colleagues on many aspects of modern therapeutics. With the rapid developments in the production of new drugs by the pharmaceutical industry, with which the British Ministry of Health is now maintaining close relations and co-operation, the pharmacist is required to keep abreast of

bly en 'n grondige kennis van die stowwe wat hy hanteer besit om sodoende in staat te wees om die farmaseutiese aspekte van die middels met die geneesheer te bespreek—veral met betrekking tot metodes van toediening en die stabiliteit van die geneesmiddels. Fabrikante het nog nooit agterweë gebly nie om die professie van advies te bedien nie!

Davis meen dat Brittanje 'n groot behoefte het aan meer praktiserende aptekers en tot 'n minder mate aan navorsers en onderwysers. Dit word besef dat hierdie farmaseutiese spesialiste universiteitsopleiding moet ontvang.

Ons in Suid-Afrika kan baie lesse put uit Brittanje se ondervinding—sommige aspekte waarvan gesaghebbend in die gemelde artikel vermeld word.

1. Davis, H. (1954): J. Pharm. Pharmacol., **6**, 761.

the stream and maintain a sound knowledge of the materials he handles, particularly to enable him to discuss with the medical practitioner the pharmaceutic aspects of the drugs, especially their modes of administration and presentation and their stability. The manufacturers have never been backward in offering advice to the medical profession!

Davis remarks that the need is being felt in Britain for a large number of recruits to the ranks of practitioner pharmacists, and for a minority of research workers and teachers. The training of these pharmaceutical specialists is recognized as being a function of the universities.

We in South Africa can gain much from British experience, some aspects of which are authoritatively presented in the article here cited.

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MORE ABOUT HAEMOGLOBINS [1]

Haemoglobin is not confined to the red cells of the higher animals, and is extremely widespread in nature. It is found in the blood of certain crustacea and even in some yeasts and moulds and in the root-nodules of leguminous plants. There are differences in the haemoglobins from different sources, and more than one variant of haemoglobin has been discovered even in a single species of animal.

Haemoglobin consists of an iron-containing portion known as haem (*Amer.* heme), or haematin, and a protein constituent known as globin. It appears that in the different haemoglobins the haem does not vary and that the differences occur in the structure of the globin and possibly also in the haem—globin linkages.

In man besides the 'normal' (A) haemoglobin of adults we have haemoglobin F, which is normal in the foetus and is found in the adult in hereditary and acquired conditions associated with anaemia; haemoglobin S, characteristic of the sickle cell and sickle-cell anaemia; and haemoglobin C, which like the S form is sometimes, though usually not, associated with symptoms of anaemia, as is also another haemoglobin—D. A 6th and 7th form have also been described in man—haemoglobin E and G.

The recognition of these different haemoglobins, the appearance of which in man is in general genetically determined, involves the use of various techniques such as electrophoresis, measurement of solubility, and spectrophotometry.[2] Differences have been found in crystallography, solubility, amino-acid composition, antigenic specificity, denaturation by alkali, spreading of mononuclear surface films, electrophoretic mobility, spectrophotometry, and oxygen dissociation curves; as well as in clinical significance.

The difference between the adult (A) and foetal (F) forms of haemoglobin have been attributed to the different sites of formation of red cells in the foetus and the adult. More work needs to be done to correlate haemopoiesis and the amount of haemoglobin F in the blood during intra-uterine life and after birth. It has been found in the blood in certain diseases, e.g. thalassaemia major (Cooley's anaemia)—large amounts in severe cases—and small amounts in other haematological disorders such as sickle-cell anaemia.

The sickle-cell trait, which is associated with haemoglobin S, has been a good deal discussed of late,[3-6] and also thalassaemia and related and interconnected groups; an excellent review has been published by White and Beaven.[7]

While some methods required for the characterization of the abnormal haemoglobins are highly specialized, others can easily be applied, and should be used in the search for variants of haemoglobin in obscure haemolytic anaemias, particularly where sickling of the red cells or features such as numerous 'target cells' are observed.

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BLOOD TRANSFUSIONS : BLOEDOORTAPPING

Doctors concerned are reminded that the Headquarters Bank of the Western Province Blood Transfusion Service at 57 Shortmarket Street, Cape Town (Tel.: 2-3174) is now open day and night and that, whilst emergencies will continue to take priority, blood can now be booked and set aside in the Bank for periods up to 24 hours at a time. It is pointed out that if this is done and the order is subsequently cancelled, no charge will be made; whereas, once the blood is sent out from Headquarters, it cannot be returned and a charge must therefore be made.

Geneeshere word daaraan herinner dat die Hoofkwartierbank van die Westelike Provinsie Bloedoortappingsdiens, Kortmarkstraat 57, Kaapstad (Tel.: 2-3174) nou dag en nag oop is en alhoewel noodgevalle nog steeds voorkeur sal geniet, bloed nou bespreek en vir tydperke van 24 uur in die bank opsy gesit kan word. Aandag word daarop gevestig dat indien sulke besprekings gekanselleer word geen gelde betaalbaar is nie, maar bloed wat alreeds afgelewer is kan nie teruggestuur word nie en betaling daarvoor moet geskied.

PHONOCARDIOGRAPHY

ITS APPLICATION TO CLINICAL MEDICINE*

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Introduction

Auscultation as a means of picking up heart sounds is mentioned by Hippocrates [1] and later, in 1628, by William Harvey,[2] but it was Laennec [3] in 1819 who first gave a precise description of the character of heart sounds. He evolved the wooden stethoscope. From then onwards interest in the mode of production of heart sounds was keen, and in 1835 the British Association for Advancement of Science [4] established a committee to elucidate the exact causes of heart sounds. In 1893 Hurthle [5] registered heart sounds graphically for the first time. He was followed a year later by Einthoven and Geluk,[6] and Frank [7] in 1904 evolved the segment capsule. Since then great strides have been made in perfecting registration of heart sounds and the causes of these sounds have been investigated experimentally and clinically with the aid of the phonocardiograph by various workers.[8-13]

Phonocardiography is the graphic registration of sounds produced in the heart and great vessels. Its chief advantage over auscultation is that:

1. Sounds can be recorded which are below the hearing threshold or inaudible owing to masking and fatigue effects on the ear from preceding loud sounds.

2. Sounds can be accurately timed in relation to the cardiac cycle.

3. It is possible to study the intensity, pitch and duration of sounds which have diagnostic significance.

By the use of microphones which transform sound vibrations into electric waves, it is possible with the aid of different types of bell and Bowles chest pieces to accentuate selected sounds and murmurs while others are attenuated. The stethoscopic microphone records sounds below the hearing threshold. It registers cardiac sounds and murmurs as they are produced in the chest wall. The logarithmic microphone registers sounds and murmurs which are normally audible, and when used with the Bowles type of chest piece can selectively accentuate high-pitched sounds and murmurs, and attenuate low-pitched ones. It is particularly useful in detecting high-pitched murmurs such as one encounters in aortic insufficiency.

Simultaneously with the heart sounds an electrocardiogram and/or a jugular phlebogram recording provide an adequate reference tracing to decide the circulatory events responsible for the sounds or murmurs. The phlebogram is particularly useful as it gives valuable information by portraying the different phases of cardiac activity. The *a* wave is due to auricular systole. The *c* wave is due to the onset of ventricular systole, and the *v* wave is due to the filling of the auricle, and its peak corresponds to the opening of the AV valves.

To evaluate the intensity of the sounds and murmurs a standard signal is provided in the machine, which has a sensitivity of 90 decibels above human hearing, and a frequency of 60 cycles per second. By comparing the two an approximate evaluation, in decibels, of the sounds and murmurs is obtained.

Over 1,500 phonocardiographic tracings have been recorded in the course of this study, and the tracings described here, are in general a reflection of the sounds and murmurs of the different cardiac conditions examined.

Two machines were used:

(1) The Cambridge Portable Electrocardiograph-Stethograph simple Trol model.

(2) The Sanborn Twin Beam Cardiette model 62.

GENESIS OF HEART SOUNDS

Heart sounds are produced by vibrations originating in the closure and opening of the valves, muscular contraction of the ventricles and the auricles, and the flow of blood through the large vessels.

Four heart sounds may be seen in the normal phonocardiogram and each may be broken down into several components.

In the *1st heart sound* 4 components are recognized. Its 1st component is represented by one or two low-frequency and low-amplitude vibrations due to 'development of tension in the ventricular muscle fibres which indicates the rise in intraventricular pressure'.[14] It is best demonstrated in the stethoscopic tracing at the apex of the heart beat. It commences at the peak of the R wave of the QRS complex of the electrocardiogram (ECG).

The 2nd and 3rd components—the valvular components—record the closure of the mitral and tricuspid valves and the opening of the semilunar valves. There is nearly always a clear separation between them. They are of high frequency and high amplitude. They end before the rise of the *c* wave of the jugular pulse, i.e. the beginning of the ejection phase.

The 4th component is considered to be due to the flow of blood in the large arteries during the phase of maximal ejection. It extends to the peak of the *c* wave of the jugular pulse.

The 2nd and 3rd components are high-frequency sounds, and are heard well with the ordinary stethoscope,

* A paper presented at the South African Medical Congress, Port Elizabeth, June 1954.

but the 1st and 4th are of low-frequency and are seldom heard.

The total duration of the 1st heart sound varies considerably—from 0·1 to 0·16 seconds.

The *2nd heart sound* also consists of 4 components. The 1st component is a low-pitched vibration and is muscular in origin, due to ventricular relaxation in commencing diastole. The 2nd component is of high amplitude and high pitch, and represents the closure of the semilunar valves. The 3rd component is composed of one or two coarse vibrations of smaller amplitude and is vascular in origin, representing blood passing through arteries. The 4th component is represented by a low vibration, and is due to the opening of the mitral and tricuspid valves. It coincides with the apex of the *v* wave of the phlebogram, and becomes audible in mitral stenosis, when it is known as the opening snap of the mitral valve. Its duration varies from about 0·08 to 0·14 seconds.

The *3rd heart sound* occurs in diastole following closely on the 2nd sound and is commonly heard in young people up to the age of 20 years. It is uncommon after the 3rd decade. If it is heard after the age of 40 years a pathological condition of the heart should be suspected, e.g. hypertensive heart disease or cardiac infarction.[15] It coincides with the descending limb of the *v* wave of the phlebogram and is thought to be caused by vibrations of the ventricular wall during its distention by the inrush of blood from the auricle in the final phase of rapid ventricular filling. Sloan *et al.*[16] found that this sound was heard in 9·8% of university students between the ages of 17 and 32 years, but that it was recorded by the stethoscopic microphone in 100% and in only 39% when the logarithmic microphone was used. My stethoscopic tracings invariably show this sound in all young people. It is best heard at the apex and with the subject lying on the left side. It can be increased by pressure on the abdomen or raising the limbs on exercise. It occurs about 0·16 to 0·24 seconds after the beginning of the 2nd sound and has to be differentiated from other sounds in diastole, e.g. opening snap, splitting of the 2nd sound and auricular sounds. It is of low frequency, differing thus in quality from the opening snap, which is higher pitched and 0·03 to 0·14 (average about 0·07) seconds distant from the 2nd sound. It is not recorded in mitral stenosis, but if present in rheumatic valvular disease it is very suggestive of mitral incompetence.[17]

The *4th or auricular heart sound* is very seldom heard. This is due to the fact that it is of very low intensity and frequency and if present is often attenuated by a loud higher-pitched 1st heart sound. With the stethoscopic microphone it is recorded in 88% of normal cases, but only in 21% with the logarithmic microphone.[12] It is caused by muscular contraction of the auricle and the passage of blood through the valvular orifice. It is situated between the P and the Q waves of the ECG, and generally 0·08 to 0·14 seconds after the peak of the P wave (Figs. 1, 2 and 3).

Apart from the ordinary heart sounds other sounds are often present, which follow each other in rapid succession, and the ear is unable to appreciate their position in the cardiac cycle. The phonocardiograph becomes extremely important and offers the best way of differentiating such sounds.

Splitting of the 1st and 2nd Sounds

The 1st heart sound may be normally or abnormally split. There is usually a distinct separation between the 2nd and 3rd components, and this separation may become audible normally. When there is asynchronous closure of the mitral and tricuspid valves, as occurs in bundle-branch block, the splitting may be abnormally wide and may be confused with extra sounds (Fig. 4).

Splitting of the 2nd heart sound is normally found in healthy children in the pulmonary area and may be accentuated by deep inspiration. A split second sound is an indication of the existence of both aortic and pulmonary vessels,[18] and is of importance in congenital heart disease.[19] When present, the 1st component is due to closure of the aortic valves and the 2nd to the closure of the pulmonary valves. In congenital heart disease or pulmonary hypertension accentuation of one or other of the components of the 2nd heart sound is of diagnostic importance. In pulmonary hypertension the pulmonary element is louder than the aortic; in pulmonary stenosis on the other hand the aortic element is louder and the tracing comprises a very long single vibration. In the aortic and apical areas the 2nd sound is usually single.

An early single or double systolic sound following immediately on the 1st heart sound is sometimes heard in the precordial area in the 3rd or 4th interspace to the left of the sternum near the apex. It is short, high pitched and clicking in nature. It may be early, middle or late in systole. This sound was first described by Gallavardin[20] in 1913. He applied to it the terms 'pseudo-reduplication of the 1st heart sound' and 'telesystolic extracardiac sound.' He believed that the sound was produced by tugging on pleuro-pericardial adhesions during systole—a conclusion arrived at from necropsy findings in 3 patients who had exhibited the sound during life. It is called the 'mid-systolic click',[21] and also '*le claquement meso-systolique pleuro-pericardiale*'.[22] It may occur in association with spontaneous mediastinal emphysema and it may be produced by injecting air in the mediastinal tissues.[21] It has no cardiac pathological significance, and its importance lies in the fact that it is sometimes mistaken for evidence of heart disease (Fig. 5).

A similar click in early systole is often noted in the pulmonary area in conditions of pulmonary hypertension and dilatation of the pulmonary artery. It is called the 'semilunar opening click',[23] and is considered to be a valuable sign in the diagnosis of enlargement of the pulmonary artery with or without pulmonary hypertension.[24] This systolic click at the base of the heart, unlike the systolic click at the apex, is therefore of diagnostic importance (Fig. 6).

Variation in the Heart Sounds

The intensity of the 1st heart sound varies with certain conditions. Those who suffer from emphysema, obesity, pericardial effusion, myocardial disease or mitral regurgitation may have decreased 1st heart sounds. Increase in intensity of the 1st sound depends upon the vigour of the ventricular systole. Thus we get loud

sounds in exercise and emotional states, and in hyperthyroidism, anaemia, rheumatic fever and hypertension.

The conditions giving rise to change in intensity of the 1st heart sound has been discussed by various authors.[25, 8, 26, 23] Dean [26] in experiments with perfused cats' hearts showed that the septal cusps of the mitral valve underwent slight upward movement during the last moments of passive filling and then sharp downward opening movement with auricular contraction. Wolferth and Margolies [23] in a study of 500 cases noted that there is a relationship between the PR interval of the ECG and the intensity of the 1st heart sound. Thus with a PR interval of 0·04 to 0·08 seconds (normal range about 0·16 to 0·18 seconds) the 1st heart sound tended to be loud. Utilizing the above observations Levine [27] explains the variation of the 1st sound on the hypothesis that when the auricles contract the AV valves are driven down into the ventricles by the rush of blood, and when the ventricles fill the AV valves float upwards to higher levels in the ventricles. If the ventricles start to contract soon after the auricles, i.e. when the PR interval is short, the leaflets are still low in the ventricular cavity and are relaxed. They snap back a considerable distance and the sound of their closure is loud. When, however, the PR interval is long the leaflets have had time to float up higher in the ventricle, the slack in the margins of the leaflets is taken up and the distance to the orifice is short, then a faint sound is produced. This is well illustrated in complete heart block, where the changing intensity of the 1st heart sound is noted with the change in the PR interval and a clinical diagnosis can be made at the bedside on auscultation (Fig. 7).

Sounds and murmurs can be modified by varying the pressure of the chest piece against the chest wall. When an open bell is used, the skin bounded by the lip of the bell acts as a diaphragm and the fleshy portion under the skin acts as a damping medium. By heavy pressure, the skin under the bell is tightened, low-pitched sounds and murmurs are attenuated and high-pitched ones stand out more clearly and distinctly. Thus heavy pressure on the chest-piece will eliminate or reduce low-pitched sounds or murmurs and bring out more clearly high-pitched sounds or murmurs (e.g. a murmur of aortic incompetence). Low-pitched sounds and murmurs (e.g. 3rd heart sounds and gallop sounds) can best be demonstrated with light pressure (Fig. 8).

Triple and Quadruple Sounds

Extra sounds are encountered in systole and diastole. When in diastole and having a canter-like quality an extra sound is known as a gallop (Fig. 9). The extra sound in systole may be due to reduplication of the first heart sound or to a systolic click. When in diastole it may be due to reduplication to the 2nd heart sound, an opening snap of the mitral valve, a 3rd heart sound or an auricular sound. Quadruple sounds heard in the cardiac cycle may be due to a combination of sounds mentioned above.

A summation gallop is the name given to a sound due to merging of the 3rd and 4th heart sounds when the ventricular rate is rapid. It is low pitched and its importance lies in the fact that it may be mistaken for a mid-diastolic murmur of mitral stenosis (Fig. 10).

MURMURS

'Murmurs' are more difficult to record than 'sounds' because generally they are of low intensity and high frequency. In addition extraneous sounds are picked up such as respiratory sounds, friction between microphone and skin or hair of the chest wall, vibrations caused by tense muscles, or thoracic movement. It is most important, in order to have a clear record, to eliminate all extraneous sounds. A quiet room, accurate apposition of the chest-piece of the microphone, and perfect relaxation of the patient, are essential. The record should be taken with respiration completely suspended in full expiration or in the optimum position in the respiratory cycle.

Mechanism of Production of Murmurs

Corrigan [28] was the first to offer an explanation of murmurs. He attached a piece of intestine to a tap and found that when the tube was constricted a bruit and a thrill were produced beyond the constricted portion provided the rate of flow was sufficiently great. Two factors are therefore necessary for the production of murmurs: (1) variation in the calibre of the vessel, and (2) velocity of the blood flow.

Bondi [29] noted that in a tube with smooth walls a murmur could be produced only when the velocity of the flow exceeded 200 cm. per second. Below that critical velocity murmurs are produced only if some roughness or any other irregularity exists in the wall of the vessel, when eddies and turbulence in the stream are produced. If, however, the rate of flow is less than 100 cm. per second no murmur is produced even in tubes with rough linings and constrictions.

The cardiovascular system lends itself to the creation of murmurs. The heart has irregular walls, membranous structures, chordae tendiniae, papillary muscles and chambers of unequal size. Eddies and turbulence are therefore liable to be formed and murmurs produced, but before a murmur can occur the blood flow must attain a certain critical level of velocity. In a normal heart with a velocity less than 200 cm. per second no murmurs are produced. When, however, the velocity of the blood flow is increased beyond the critical level, e.g. in increase in pressure-gradients between chambers, in increase of heart rate during exercise, in hyperthyroidism etc., a murmur is produced. We can thus have functional and organic murmurs. The detection of a murmur, therefore, does not justify the conclusion that the heart is diseased and, contrariwise, the absence of a murmur does not necessarily mean that the heart is not diseased.

Murmurs appear during systole and diastole. It is well known that whereas diastolic murmurs generally denote some form of heart disease, systolic murmurs are frequently heard in healthy individuals without heart disease.

Systolic Murmurs

Levine [27] defines a systolic murmur as 'a sound, however faint, that continues well beyond the 1st heart sound and extends at least through the first quarter or third of systole'. The assessment of such a murmur

on a functional or organic basis is often extremely difficult. MacKenzie minimized the significance of systolic murmurs. Masters [30] advises that 'a loud systolic murmur at the apex should be considered organic until it can be proved otherwise'.

Freeman and Levine [31] classified systolic murmurs into 6 grades. Grade 1 is considered as the faintest murmur, which is generally not heard until one listens very carefully for a short while so as to get the ear tuned to its presence. Grade 2 is a slight murmur heard immediately the stethoscope is placed on the chest wall. Grade 3 is a moderate and grade 4 a loud murmur. Grade 5 is loud and harsh but the stethoscope must be on the chest wall for the murmur to be heard. Grade 6 is extremely loud, harsh and rasping and can be heard with the stethoscope just removed from the chest wall. Grades 1 and 2 are considered as functional and 3 to 6 as organic. Appreciation of different grades comes with auscultatory experience and a rough measure of the nature of the murmur, whether organic or innocent, can be made. In assessing a murmur, however, factors such as anaemia, hyperthyroidism, tachycardia, fever and hypertension should be ruled out before an organic basis for fainter murmurs is given.

Attempts have been made to establish the nature of a murmur by the phonocardiogram. Evans [32] assesses systolic murmurs as innocent when they occur in mid-systole or in late systole. Using limb lead 2 of the ECG as a reference tracing, he found that when a line is drawn from the S line to touch the simultaneous tracing of the phonocardiograph the innocent murmur commences a short distance after the S line but the organic murmur commences at the S line. This relationship, however, has been disproved by Cowen and Parnum,[33] who could find no clear-cut distinction between an innocent and an organic murmur on such relationship to the S line. My own tracings bear out the contentions of the latter authors. Harris and Freeman [34] have tried to distinguish innocent from organic systolic murmurs on the basis of 'low frequency vibrations and uniform wave pattern' (Fig. 11) as against a murmur of 'chaotic mixture of amplitudes and frequencies' (Fig. 12). While my tracings suggest this possibility, in a great number of instances it is quite impossible to assess the nature of the murmur on the phonocardiograph. A systolic murmur of grade 3 intensity and upwards is more likely to be organic. Such intensity is often manifested in the phonocardiograph by high amplitude of the vibration, commencing early in systole, and even following on the 1st heart sound and gradually decreasing in amplitude. However, it is important to remember that murmurs of slight intensity registered at one period in a heart which is considered to be perfectly healthy may at a later stage prove to be organic.

MITRAL STENOSIS

Durozier [35] in 1862 described the sound in mitral stenosis as 'ffout-tata-rou'. The 'ffout' corresponding to the presystolic crescendo murmur followed by a loud snapping 1st heart sound, 'tata' to the duplication of the 2nd heart sound, and 'rou' to the diastolic rumble. Rouches [36] in 1888 introduced the term '*claquement d'ouverture de la mitrale*' (the opening snap of the mitral valve) to explain the 2nd element of the 2nd heart sound. Since then studies made by various workers [23, 37,39] have confirmed the accuracy of the above observations.

As a variety of acoustic phenomena occur in both systole and diastole the phonocardiogram is extremely important for an accurate differentiation of the various events. The 1st heart sound is generally extremely loud, snapping and delayed. Normally the maximal vibration of the 1st heart sound starts within 0·06 seconds of the onset of the QRS. In mitral stenosis the maximal vibration commences 0·07 seconds or more later.[38] Marked accentuation of the 1st heart sound is important 'as a reassuring sign that stenosis is a predominant lesion'.[40] This degree of intensity of the 1st heart sound can be explained on the basis of the position of the valves in the ventricular cavity at the moment of ventricular contraction. With stenosis the ventricles fill slowly; hence the cusps are low when the ventricles contract and the 1st sound is therefore loud (Fig. 13). In mitral incompetence and in the presence of calcified valves the 1st heart sound is usually soft. In incompetence the ventricles are filled rapidly and the valves are high, hence the sound is soft at the moment of closure. With calcification the valves hardly move; hence the sound is soft. In auricular fibrillation, when diastole is short the 1st heart sound is loud, when prolonged it is soft. A systolic murmur usually follows the 1st heart sound. Generally a loud pan-systolic murmur at the apex is associated with mitral incompetence, but in our experience the absence of such a murmur does not necessarily exclude incompetence (Fig. 14). Mounsey,[41] however, found that where an apical systolic murmur was absent on auscultation and on the phonocardiograph no incompetence was felt at mitral valvotomy.

The Opening Snap

This is now considered to be the most important indication of mitral stenosis. The sound is due to the sudden stretching of the stenosed valves with the inrush of blood from the left auricle.[42] It is high pitched and is usually heard in the mitral area inside the mid-clavicular line. It is also heard in the pulmonary area or along the left sternal border. The time interval between the beginning of the 2nd element of the 2nd heart sound and the opening snap varies from 0·03 to 0·14 seconds; the average is about 0·07 seconds. It is seen opposite the peak of the *v* wave of the phlebogram. It immediately precedes the mid-diastolic murmur, or there may be a small gap between it and the murmur. In cases where there is gross calcification of the mitral valves the opening snap may be absent. It is not abolished in auricular fibrillation, where its distance varies with the preceding RR interval. Where the preceding diastolic interval is increased the interval before the opening snap is greater than when the preceding interval is short.[41] The opening snap may be the earliest indication that mitral stenosis is in progress (Fig. 15). Levine and Harvey [43] relate how in 'some rheumatic cases observed over the course of years, at first only this opening snap could be heard without any detectable murmur in diastole. Then as time went on it became clear that a

slight and later a more pronounced diastolic rumble developed with this 3rd heart sound'.

The opening snap has to be differentiated from a split 2nd heart sound. The average interval between the beginning of the 2nd component of the 2nd heart sound and the beginning of the 3rd component is generally about 0·03 seconds or a little more in a split sound, whereas the opening snap averages about 0·07 seconds. It has also to be differentiated from a 3rd heart sound. The latter is lower pitched and its distance from the commencement of the 2nd heart sound is between 0·16 to 0·24 seconds. The peak of the *v* wave of the jugular pulse tracing is opposite the opening snap (Fig. 16), whereas the 3rd heart sound falls along the descending limb of the *v* wave (Fig. 2). It is rare to find a 3rd heart sound in mitral stenosis. We have often noted it in mitral incompetence.

The diastolic rumble generally starts after the opening snap but may be delayed and start after a short pause. It usually becomes more intense in the region of the 3rd heart sound owing to the more rapid flow of blood into the ventricle. It may fade in a diminuendo fashion and stop short of the 1st heart sound (Fig. 17), or it may continue in a crescendo manner to the 1st heart sound, giving rise to what is known as presystolic accentuation. It is of low frequency and is therefore often missed on auscultation. The bell chest-piece lightly applied will assist in its detection and the stethoscopic microphone is used for its registration. While a mid-diastolic murmur is generally considered to be an indication of mitral stenosis, Bland *et al.*[44] have noted that it may be present in a dilated ventricle and disappear as the heart returns to normal size. We have noted such diastolic murmurs disappear in cases which have been successfully operated for patent ductus arteriosus.

The Presystolic Murmur

When this murmur is noted at the apex of the heart it is considered as pathognomonic of mitral stenosis. It may be found alone or in association with a mid-diastolic murmur. It may continue in a crescendo manner to the 1st heart sound (Fig. 16), or there may actually be a slight pause between the mid-diastolic murmur and the presystolic murmur (Fig. 13). It may be the earliest sign of mitral stenosis. It is associated with auricular systole during which the velocity of the blood flow through the stenosed mitral valve is increased. When, however, the auricular contraction is feeble the velocity of the blood flow through the valve is diminished and the presystolic murmur becomes inaudible.

Mitral stenosis may be diagnosed erroneously on the basis of an acoustic impression of a presystolic murmur. The latter may be simulated by an intensely loud 1st heart sound, or by a duplicated 1st heart sound especially when the 2nd valvular component is louder than the first, thus giving the acoustic impression of a crescendo-like murmur (Fig. 18).

Valvotomy in well-selected cases, when successful, gives remarkable clinical improvement, but the auscultatory post-operative findings in the vast majority of cases still show the pre-operative sounds and murmurs, but reduced in intensity (Fig. 19). It is likely, however, that the persistence of the murmurs may be due to the fact that the valves have not been split widely enough at operation. I am now informed that attempts are being made to split the valves wider. The results, assessment, contra-indications and limitations of valvotomy have been fully and ably discussed in this *Journal*.[45-48]

Tricuspid Stenosis and Incompetence

The murmurs in this condition are generally similar in nature and configuration to those encountered in mitral stenosis, but are heard best along the left and right sternal borders. The incompetent systolic murmur becomes more intense in full inspiration. The condition is usually associated with mitral stenosis (Fig. 20).

AORTIC STENOSIS AND REGURGITATION

The systolic murmur in aortic stenosis takes on a characteristic configuration. It is symmetrical, crescendo-decrescendo, starts late in systole, becomes intense in mid-systole, and ends before the 2nd heart sound. It takes on a diamond-shaped configuration. Its constancy is so invariable that it is extremely valuable as a diagnostic sign (Fig. 21). Occasionally it has to be differentiated from the systolic murmur of mitral incompetence in the mitral area. The diamond-shaped murmur recorded in the mitral area will give the answer (Fig. 22).

Basal Diastolic Murmurs

The murmurs of aortic and pulmonary regurgitation result from the backflow from the aortic and pulmonary vessels during diastole. They usually follow immediately on the 2nd heart sound, are blowing in character, high pitched and diminuendo throughout diastole. Wells *et al.*[49] have shown that the murmur may be crescendo-decrescendo in configuration and if the 2nd heart sound is single in configuration there may be a slight gap between the 2nd sound and the commencement of the murmur. When the 2nd sound is split there may be a few vibrations between the two components and the murmur therefore commences with the 1st component, thus giving the crescendo-decrescendo tracing. The early diastolic murmur is often very faint and is therefore frequently missed on auscultation. It is best recorded with the logarithmic microphone and with the Bowles chest piece (Fig. 23).

It is difficult to differentiate on the phonocardiograph between aortic incompetence and pulmonary incompetence. The clinical evidence of a collapsing pulse is of assistance. An early diastolic murmur which is best picked up at the lower end of the sternum is likely to be due to aortic incompetence. When the murmur is located in the pulmonary area it is more likely to be pulmonary incompetence.

A *musical diastolic murmur* due to aortic regurgitation is occasionally met with. Bellet *et al.*[50] have described such murmurs and have attributed them to retroversion of the aortic cusps which, in their opinion, is almost invariably associated with syphilis. The musical quality of the sound has suggested various descriptive terms, such as cooing dove, cuckoo clock, humming of a top or buzzing of a saw. The murmur has a frequency of 200 cycles per second. It is more intense and higher in pitch than a systolic murmur. It is very characteristic in

its configuration. It commences at the 2nd heart sound, which is indistinct and takes on a decrescendo character, fading as it approaches the 1st heart sound, which is hardly visible (Fig. 24).

A *musical systolic murmur* was encountered in a case which had recovered from subacute bacterial endocarditis. It is recorded in Fig. 25 and probably due to rupture of one of the mitral valves.

(A *musical extracardiac murmur* was heard in a case which presented itself as a problem regarding an extra sound in systole and a musical short late systolic murmur. The phonocardiograph shows a soft split 1st heart sound, a late systolic click and a very short late murmur the configuration of which shows a frequency typical of a musical murmur. Because of its association with the systolic click it was considered to be extracardiac in origin, probably pleuro-pericardial—Fig. 26).

CONGENITAL HEART DISEASE

The advent of cardiac surgery in recent years has changed the outlook of congenital heart disease completely. Phonocardiography plays an important part in assessing the nature of the lesion and assists greatly in the final diagnosis.

Patent Ductus Arteriosus. The murmur in this condition, likened in description to machinery, is continuous in character and is best heard in the pulmonary area. It may be heard at the back or along the left infraclavicular region. Starting late in systole it increases in intensity to the 2nd heart sound, which it envelops, and continues in a decrescendo manner throughout most or all diastole. It is most intense in systole (Fig. 27). A mid-diastolic murmur is sometimes noted in the mitral area (Fig. 28). It is suggested that this murmur is due to rapid ventricular filling of an enlarged left ventricle. It starts with the 3rd heart sound and is of lower frequency. After successful ligation the continuous murmur completely disappears, as does the mid-diastolic murmur. Sometimes a faint systolic murmur remains (Fig. 29).

Reversed Patent Ductus Arteriosus. A number of cases of patent ductus have been encountered which did not show the usual features of a continuous murmur. These cases were associated with cyanosis and invariably with pulmonary hypertension as proved by cardiac catheterization. The flow of blood instead of from the aorta to the pulmonary artery is shunted through the patent ductus in the reverse direction. The phonocardiograph shows the following features. The 1st and 2nd heart sounds are distinct and loud. The systolic murmur is early, short and decrescendo. A diastolic murmur follows on the 2nd heart sound, is mildly decrescendo and is prolonged, occupying practically the whole of diastole. Some of these cases were successfully ligated by Mr. D. Adler, and the murmurs, though still noticeable on the post-operative phonocardiogram, are reduced in intensity. Clinically the patients felt very much better (Fig. 30).

A patent ductus has to be differentiated from (1) arterio-venous fistula (Fig. 31), (2) venous hum (Fig. 32), and (3) the continuous murmur of ruptured sinus of Valsalva (Fig. 33).

The configuration of an arterio-venous fistula is similar to that of a patent ductus. It is usually very intense over the fistula and is transmitted over a wide area. Bone transmission is usually well marked, and, depending upon where the fistula is situated, the murmur can be heard at a distance in the skull, elbow etc. The mechanism of the murmur in these conditions follows the explanation of murmurs in general. There is an abrupt change in vessel diameter, an increase in the velocity of the blood flow, and an extreme gradient from a high-pressure level in the artery to the low pressure in the vein, giving rise to rapid flow through the fistula with consequent vibrations and eddies.

Pulmonary Stenosis. In pulmonary stenosis the murmur is registered best in the 2nd left interspace. The 1st sound is usually soft and the 2nd sound is single and represented by the aortic component. The systolic murmur is diamond-shaped, with vibrations of high frequency and high amplitude. It starts a short distance from the 1st heart sound, develops a crescendo configuration with maximum intensity beyond mid-systole, and reaches the 2nd heart sound in a decrescendo manner (Fig. 34). In some instances the systolic murmur after reaching the 2nd heart sound extends beyond it into diastole for a short distance. This early diastolic murmur is considered to be due either to post-stenotic dilatation of the pulmonary artery or to 'delay of termination of right ventricular systole, beyond the time of closure of the aortic component of the 2nd sound'[51] (Fig. 35).

In infundibular stenosis the murmur starts early in systole immediately after the 1st heart sound and ends a considerable distance before the 2nd sound. It is best located in the 4th left interspace (Fig. 36).

In mild pulmonary stenosis the 2nd sound is split, with the pulmonary component more accentuated than the aortic component. The systolic murmur is diamond-shaped and occupies practically the whole of systole (Fig. 37).

Ventricular Septal Defect. In this condition the systolic murmur is diamond-shaped and may spread over the whole of systole. The 2nd sound is split, the aortic component being of higher amplitude (Fig. 38).

Auricular Septal Defect. Here the 1st sound is broad and split and the 2nd sound is widely split, with the aortic component a little more intense, and is of high amplitude. The systolic murmur commences at the 1st heart sound and may stop short before the 2nd heart sound. A mid-diastolic murmur is often noted in the mitral area (Fig. 39).

Tetralogy of Fallot. The systolic murmur, when present, is found in the 2nd left interspace, is diamond-shaped, and is similar to the murmur noted in pulmonary stenosis (Fig. 40).

Coarctation of the Aorta. In this condition the 1st and 2nd heart sounds are usually well marked. The configuration of the systolic murmur depends on the site over which the microphone is placed. If it is over the stenosed area the murmur is diamond-shaped (Fig. 40). When the microphone is placed beyond the coarctation along the lower border of the sternum or at the back, the murmur is late in systole and is plateau-shaped (Fig. 42).

Sir James MacKenzie is reported to have said that the stethoscope should be thrown away because it does more harm than good. Since his time great strides have been made in cardiology and the value of auscultation has become fully established. The human ear, however, has inherent limitations. It is sensitive to vibrations whose frequencies lie between 20 and 20,000 cycles per second and, as the vibrations of heart sounds vary between 5 and 650 cycles per second, some cardiac vibrations become inaudible or are only heard by very keen and able observers. One can therefore appreciate the importance of phonocardiography, which registers sounds and murmurs as they are produced in the chest wall. Our cardiac clinic appreciated this important fact and a phonocardiographic department was established at its very inception. We have found phonocardiography extremely helpful in diagnosis of all sorts of cardiac conditions. It has given members of the staff confidence. It has been of great help in timing of heart sounds both normal and abnormal. It has taught us to appreciate split sounds, 3rd heart sounds, gallops, opening snaps. It has helped us to avoid pitfalls in diagnosis. It has proved of value where sounds are inaudible owing to masking and fatiguing effects on the ear from previous loud sounds. It has proved of inestimable value in mitral stenosis and it has saved many a patient who has been made a chronic cardiac invalid by erroneous auscultatory interpretation of sounds and murmurs. The pattern of vibrations of pulmonary and aortic stenosis is classic and is therefore very useful in the diagnosis of these conditions. It has proved of great value in congenital heart disease, where the configuration of murmurs and splitting of the 2nd heart sound are of diagnostic importance. The study of various patterns of tracings with systolic and diastolic vibrations has given us a better understanding of cardiology and its problems. Lastly, phonocardiography provides a permanent and lasting record of sounds and murmurs which by comparison at a later stage may prove to be of great prognostic value.

Phonocardiography has a great future. Not only does its usefulness lie in diagnostic assistance but it can and should be of value in teaching students to appreciate sounds and murmurs, for nothing is more valuable in clinical medicine than a visual appreciation of signs.

I am extremely grateful to Dr. B. van Lingen, head of the cardiology clinic, and Dr. M. McGregor and Dr. J. Gear for their encouragement and co-operation and much helpful advice; to Miss J. Whidborne, B.Sc., Mr. A. M. Shevitz and Mr. P. J. Shreve for their technical help; and to Mr. David Adler for his advice and co-operation. Prof. G. A. Elliott I wish to thank for his encouragement, reading of the manuscript and most useful suggestions.

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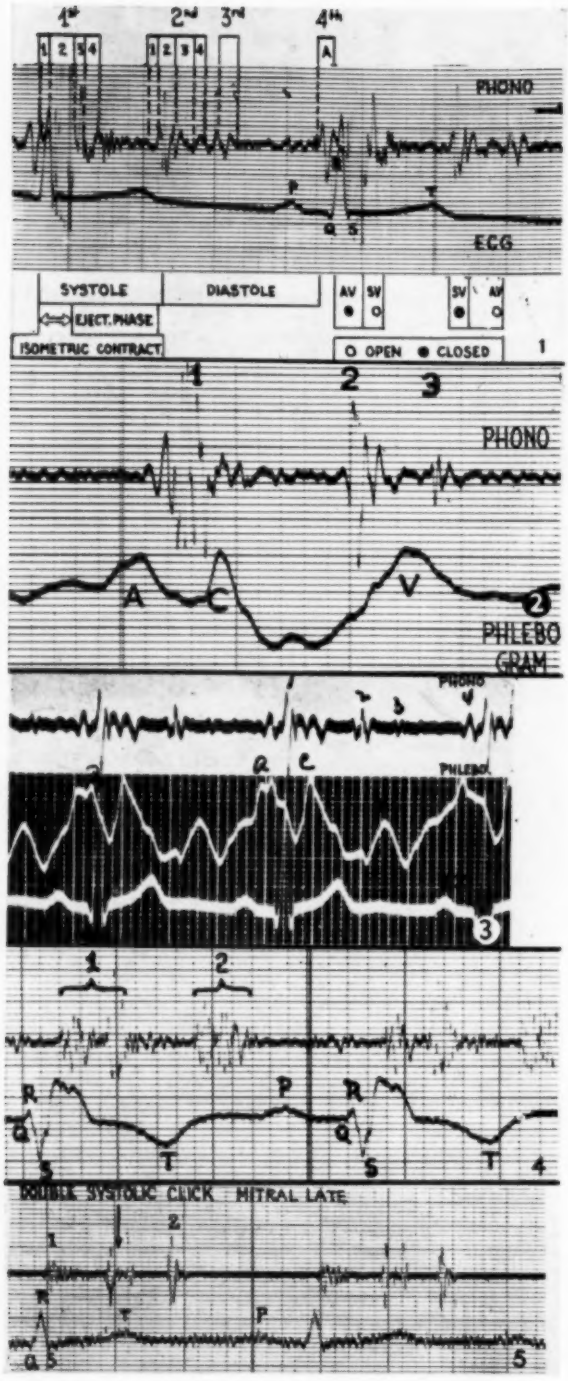
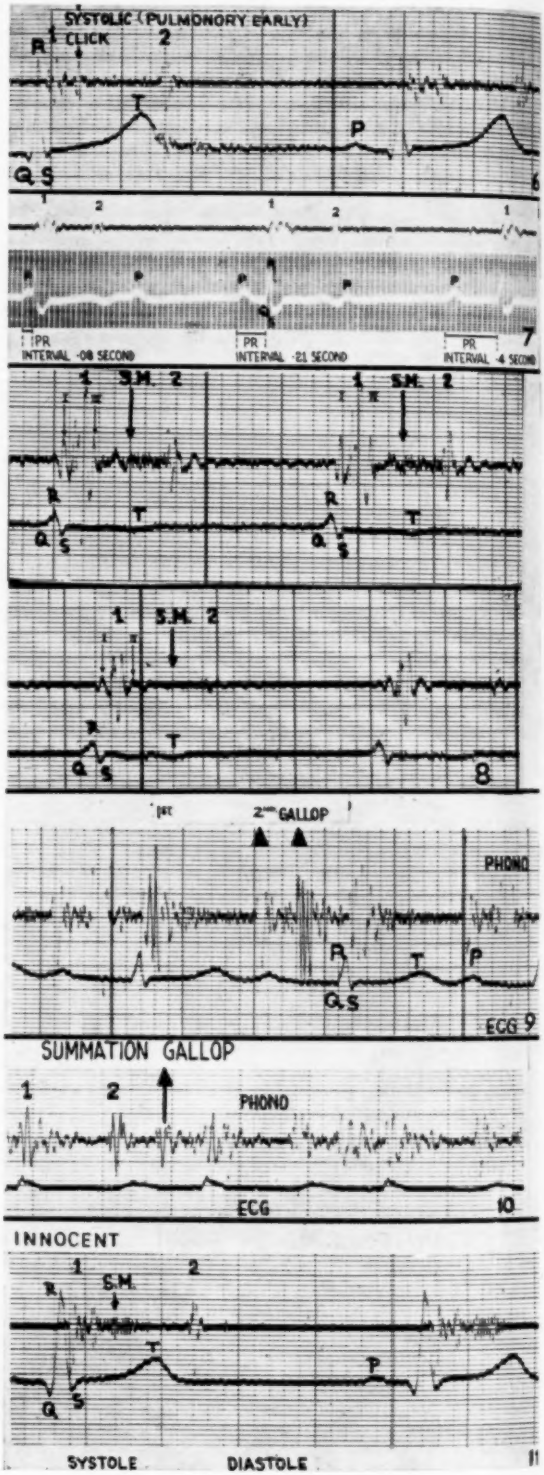
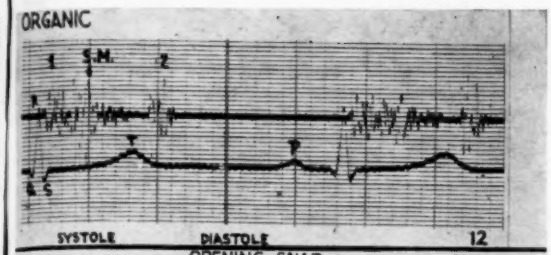
Fig. 1. Stethoscopic tracing. Normal phonocardiogram with simultaneous ECG showing the 4 heart sounds with the different components of the 1st and 2nd heart sounds. AV=auriculoventricular valves. SV=semilunar valves. A=auricular sound.
Fig. 2. Stethoscopic tracing. Normal phonocardiogram with simultaneous jugular phlebogram. Valvular component of the 1st sound ends before rise of c wave, apex of v wave opposite the 4th component of the 2nd heart sound and the 3rd heart sound opposite the descending limb of the v wave.
Fig. 3. Stethoscopic tracing. Simultaneous tracing of phonocardiogram, phlebogram and ECG.
Fig. 4. Stethoscopic tracing. Wide splitting of the 1st and 2nd heart sounds in right bundle-branch block.
Fig. 5. Logarithmic tracing. Late double systolic click in mitral area in a case of tumour of a lung.


1st
2nd
3rd
4th
PHONO
ECG
SYSTOLE
DIASTOLE
EJECT. PHASE
ISOMETRIC CONTRACT
O OPEN ● CLOSED
PHONO
A
C
V
PHLEBO GRAM
PHLEBO.
DOUBLE SYSTOLIC CLICK MITRAL LATE


SYSTOLIC (PULMONARY EARLY) CLICK
PR INTERVAL .08 SECOND
PR INTERVAL .21 SECOND
PR INTERVAL .4 SECOND
S.M.
1st
2nd GALLOP
PHONO
ECG
SUMMATION GALLOP
INNOCENT
SYSTOLE
DIASTOLE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1 S.M. 2
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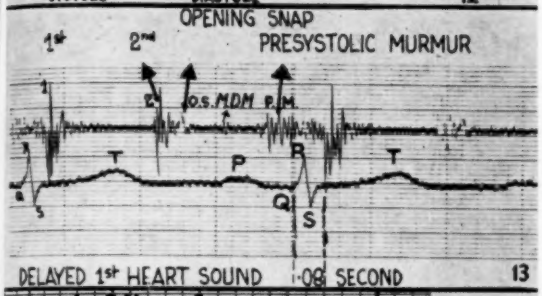
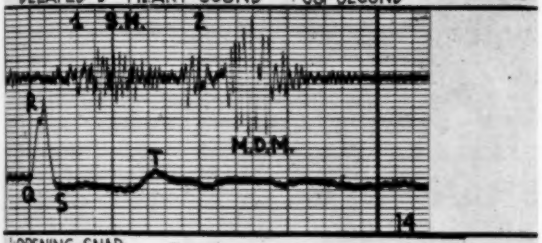
P
Q S
SYSTOLE DIASTOLE
12


OPENING SNAP
1st 2nd PRESYSTOLIC MURMUR
O.S. M.D.M. P.M.
T
P
R
Q
S
DELAYED 1st HEART SOUND .08 SECOND
13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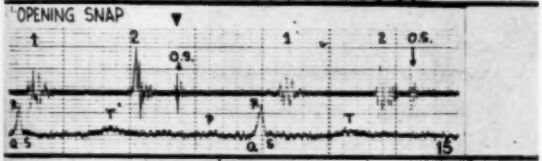
M.D.M.
Q S
14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1 2 O.S.
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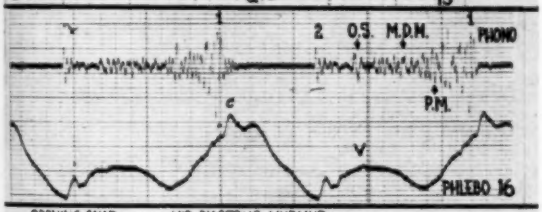
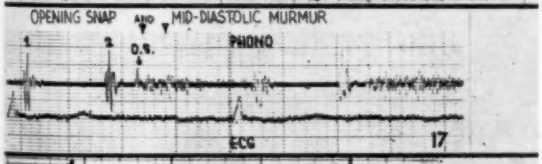
Q S
15


1 2 O.S. M.D.M. 1 PHONO
P.M.
C
V
PHLEBO 16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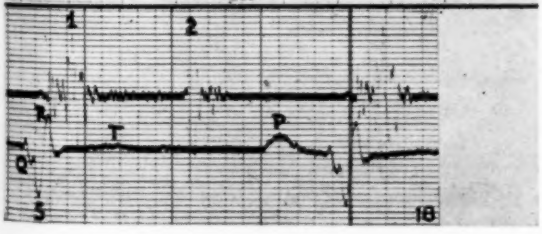
1 2 O.S. PHONO
ECG
17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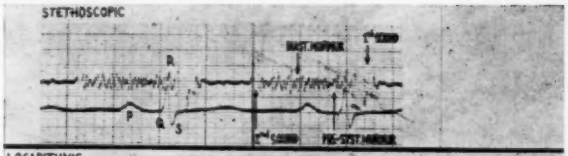
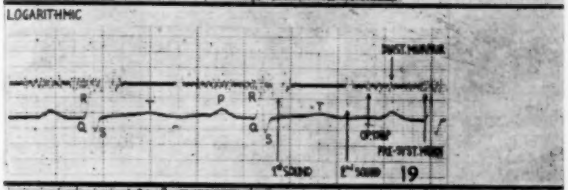
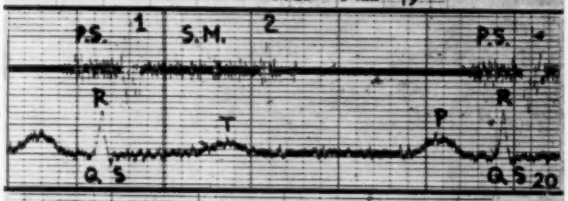

STETHOSCOPIC
R
P
Q S
1st SOUND
PRE-SYST. MURMUR


LOGARITHMIC
DIAST. MURMUR
R
T
P
Q S
OPENING SNAP
PRE-SYST. MURMUR
1st SOUND
1st SOUND
19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R
T
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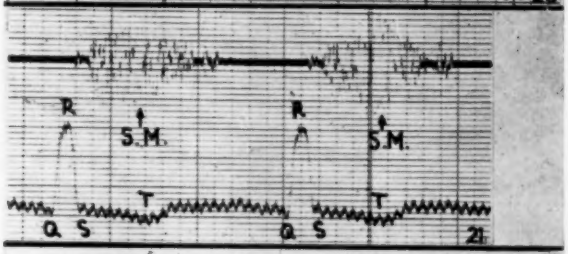
Q S
Q S
20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S.M.
R
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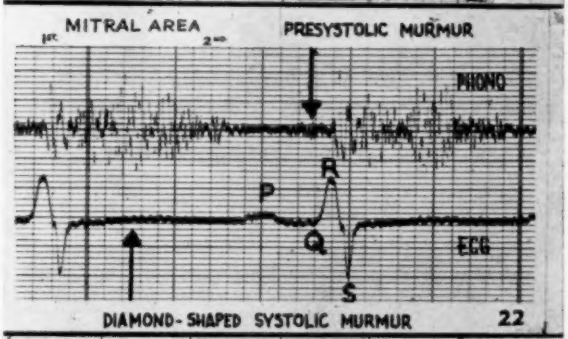
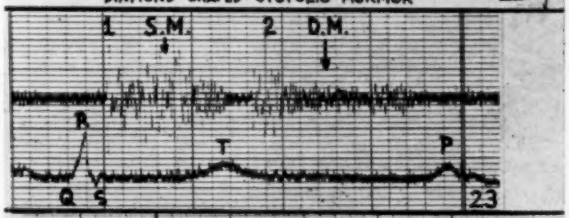
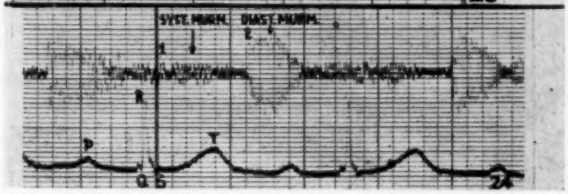
Q S
Q S
21


1st MITRAL AREA 2nd PRESYSTOLIC MURMUR
PHONO
R
P
Q
S
ECG
DIAMOND-SHAPED SYSTOLIC MURMUR
22


1 S.M. 2 D.M.
R
T
P
Q S
23


SYST. MURM. DIAST. MURM.
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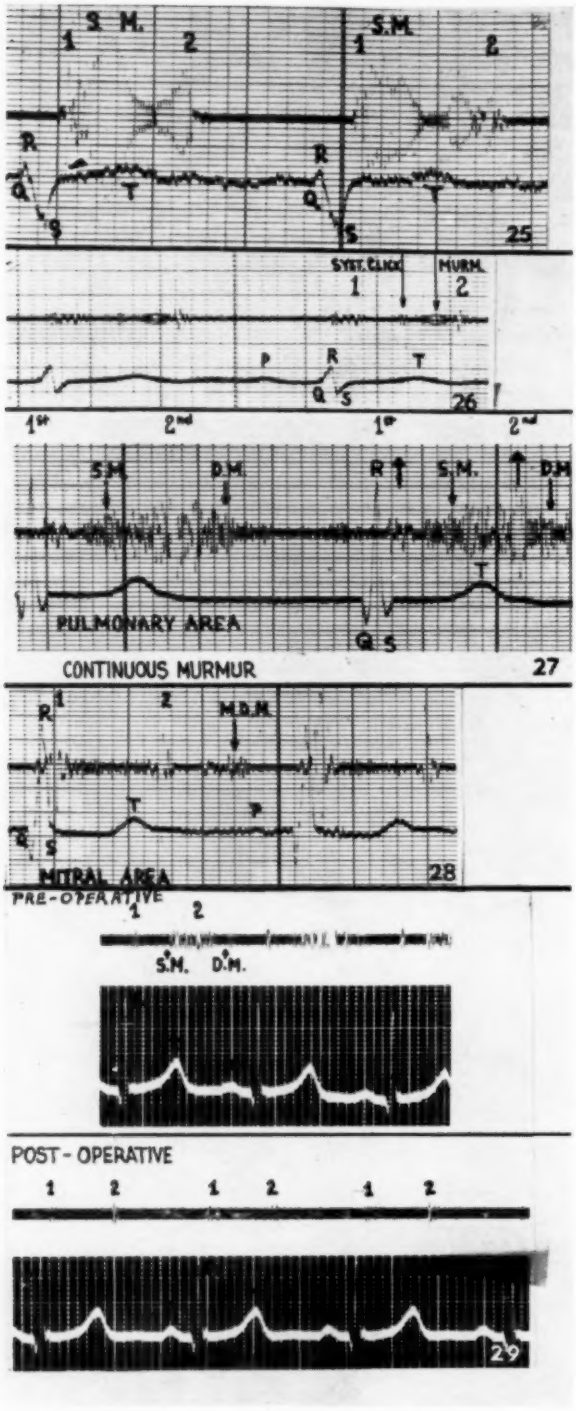
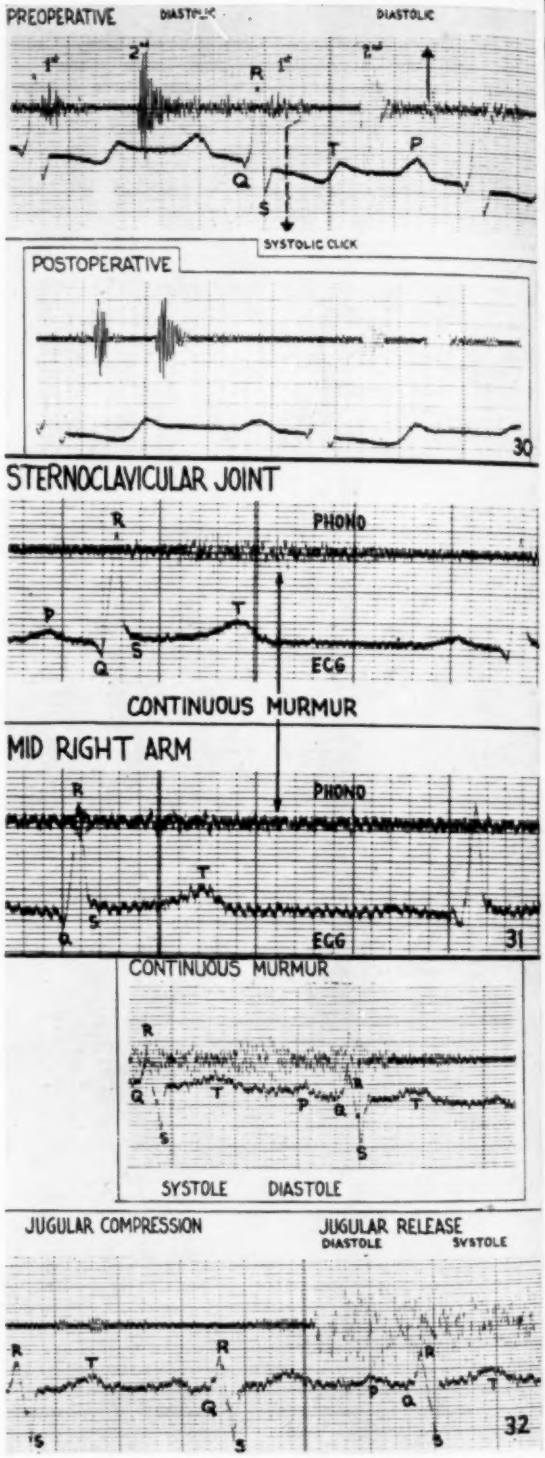
2
R
P
T
Q S
24


S.M.
S.M.
25
SYST. CLICK
MURM.
26
1st
2nd
1st
2nd
S.M.
D.M.
S.M.
D.M.
PULMONARY AREA
CONTINUOUS MURMUR
27
M.D.M.
MITRAL AREA
28
PRE-OPERATIVE
S.M.
D.M.
POST-OPERATIVE
29


PREOPERATIVE
DIASTOLIC
DIASTOLIC
SYSTOLIC CLICK
POSTOPERATIVE
30
STERNOCLAVICULAR JOINT
PHONO
ECG
CONTINUOUS MURMUR
MID RIGHT ARM
PHONO
ECG
31
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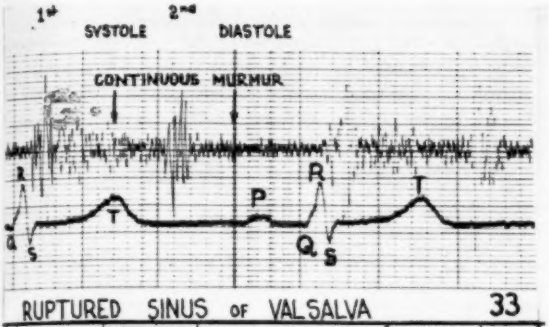
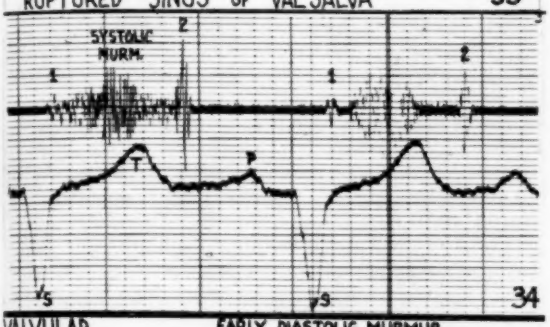
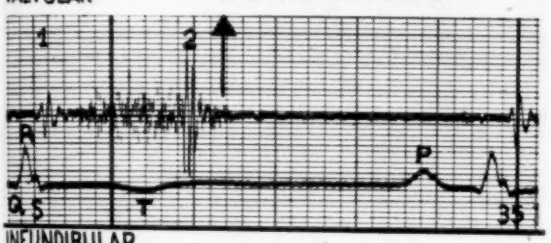
SYSTOLE
32



RUPTURED SINUS OF VALSALVA 33



VALVULAR EARLY DIASTOLIC MURMUR



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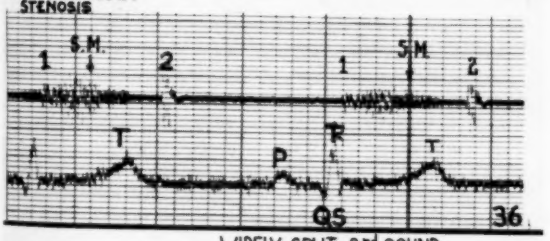
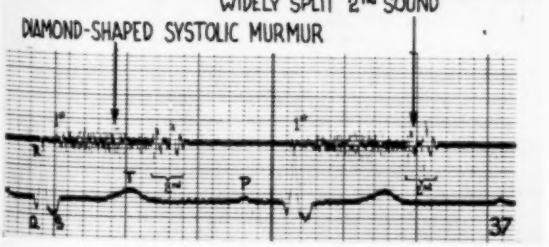
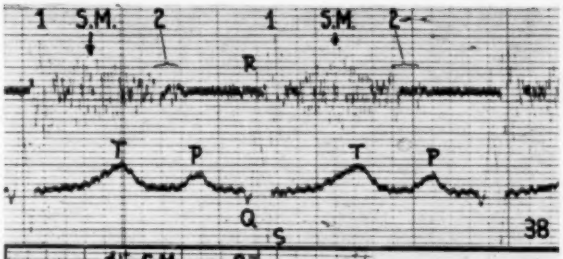
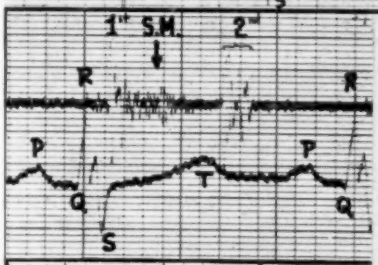


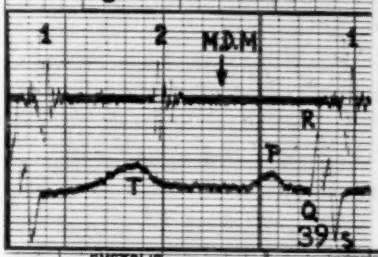
WIDELY SPLIT 2nd SOUND

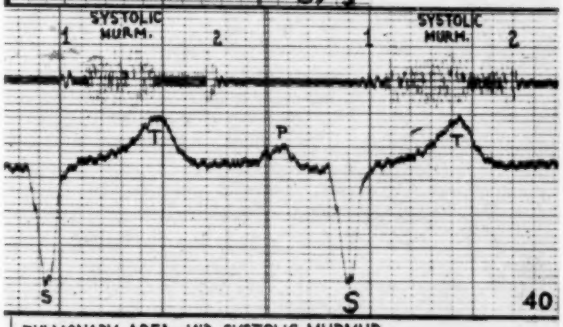
DIAMOND-SHAPED SYSTOLIC MURMUR



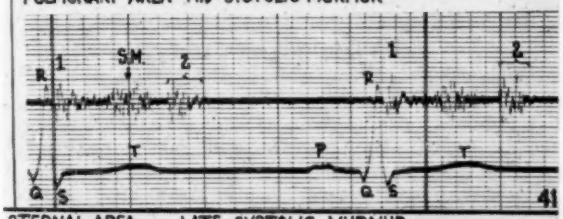








PULMONARY AREA MID-SYSTOLIC MURMUR



STERNAL AREA LATE SYSTOLIC MURMUR

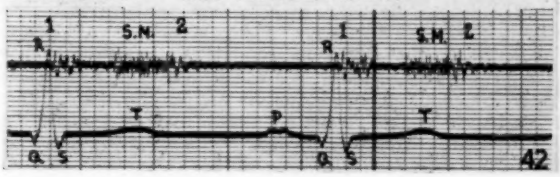


Fig. 6. Logarithmic tracing. Early systolic click in pulmonary area in a case of pulmonary dilatation and hypertension.
Fig. 7. Stethoscopic tracing. Changing intensity of the 1st heart sound in complete heart block. With P.R. interval of 0·08 seconds the duration and amplitude are 0·13 seconds and 11 mm.; with P.R. interval of 0·21 seconds the duration and amplitude are 0·08 seconds and 9·5 mm.; and with an interval of 0·4 seconds the duration and amplitude are 0·1 seconds and 8·5 mm.
Fig. 8. Changing sounds and murmurs with varying pressure. Upper tracing, light pressure. The 1st heart sound is broad and loud with 1st and 4th low-intensity components high in amplitude and the systolic murmur showing low-frequency and high-frequency vibrations. In the lower tracing with heavy pressure the 1st and 4th components of the 1st heart sound are attenuated, and only high-frequency and low-amplitude vibrations are noted in systole.
Fig. 9. Gallop rhythm. The extra sound in diastole had a canter-like quality.
Fig. 10. Summation gallop. The sound in diastole gave the impression of a rumbling diastolic murmur. The tracing shows summation of 3rd and 4th heart sounds.
Fig. 11. Innocent systolic murmur. Note low-frequency vibrations and uniform pattern.
Fig. 12. Organic systolic murmur. Note vibrations of irregular mixture of amplitudes and frequencies.
Fig. 13. Mitral stenosis. Note delayed 1st heart sounds, opening snap, presystolic murmur and faint low-grade diastolic murmur (M.D.M.). Note slight pause between diastolic and presystolic murmurs.
Fig. 14. Mitral incompetence and mitral stenosis. Note loud pan-systolic murmur, soft 1st heart sound and loud mid-diastolic murmur (M.D.M.).
Fig. 15. Mitral stenosis. Note opening snap 0·13 seconds from commencement of valvular component of 2nd sound. It was the only indication of mitral stenosis. The 1st heart sound delayed 0·08 seconds.
Fig. 16. Mitral stenosis. Opening snap (O.S.) opposite the v wave of the phlebogram, followed by a mid-diastolic murmur (M.D.M.) and with presystolic accentuation (P.M.). Note slight pause between opening snap and murmur.
Fig. 17. Mitral stenosis. Note delayed 1st heart sound 0·07 seconds, opening snap (O.S.) and mid-diastolic murmur fading towards 1st heart sound.
Fig. 18. Split 1st and 2nd heart sounds gave acoustic impression of mitral stenosis. The 2nd valvular component of the 1st sound is louder and gave the impression of a crescendo presystolic murmur and the 2nd valvular component of the 2nd heart sound gave the impression of an opening snap.
Fig. 19. Mitral stenosis. Post-valvotomy. Note presence of murmurs including opening snap (O.S.) in logarithmic tracing.
Fig. 20. Tricuspid stenosis. Note pan-systolic murmur (S.M.) and presystolic murmur (P.S.).
Fig. 21. Aortic stenosis. Note very soft 1st heart sound and diamond-shaped configuration of the systolic murmur (S.M.).
Fig. 22. Aortic stenosis and mitral stenosis. Mitral area. Note diamond-shaped configuration of the systolic murmur (S.M.) and the presystolic murmur (P.S.) and loud 1st heart sound of mitral stenosis.
Fig. 23. Aortic stenosis and aortic incompetence. Aortic area. Note diamond-shaped systolic murmur (S.M.) and early prolonged diastolic murmur (D.M.).
Fig. 24. Musical diastolic murmur (cooing dove). Note configuration and high frequency and high amplitude.
Fig. 25. Musical systolic murmur (S.M.) following subacute bacterial endocarditis.
Fig. 26. Extra-cardic musical murmur and systolic click.
Fig. 27. Patent ductus arteriosus. Pulmonary area. Note typical continuous murmur. Systolic louder than the diastolic murmur.
Fig. 28. Patent ductus. Mitral area. Systolic murmur only. Note mid-diastolic murmur (M.D.M.).
Fig. 29. Patent ductus. Upper tracing pre-operative; note continuous murmur. Lower tracing post-operative; note disappearance of murmur.
Fig. 30. Reversed patent ductus, proved by catheterization and operation. Upper tracing; note systolic click (indication of pulmonary hypertension), soft systolic murmur and loud 2nd heart sound with decrescendo diastolic murmur. Lower tracing post-operative; note disappearance of murmur.
Fig. 31. Arterio-venous fistula. Upper tracing, continuous murmur. Lower tracing, radiation of murmur along bone to elbow.
Fig. 32. Venous hum. Upper tracing, continuous murmur, diastolic louder than systolic. Lower tracing with jugular compression, note disappearance of murmur and reappearance with release of jugular vein.
Fig. 33. Ruptured sinus of Valsalva. Note continuous murmur.
Fig. 34. Pulmonary stenosis. Note diamond-shaped systolic murmur and single 2nd heart sound.
Fig. 35. Pulmonary stenosis, valvular type. Note systolic murmur reaches 2nd heart sound. Early diastolic murmur.
Fig. 36. Pulmonary stenosis, infundibular type. Note early diamond-shaped systolic murmur and single 2nd heart sound.
Fig. 37. Pulmonary stenosis, mild degree. Note diamond-shaped systolic murmur and widely split 2nd heart sound.
Fig. 38. Ventricular septal defect proved by catheterization. Note diamond-shaped systolic murmur and widely split 2nd heart sound.
Fig. 39. Auricular septal defect, proved by catheterization. Upper tracing, pulmonary area; note early systolic murmur and split 2nd heart sound. Lower tracing, mitral area; note mid-diastolic murmur (M.D.M.).
Fig. 40. Tetralogy of Fallot. Pulmonary area. Note diamond-shaped systolic murmur.
Fig. 41. Coarctation of aorta. Over coarctation murmur is diamond-shaped. Split 2nd sound.
Fig. 42. Coarctation of aorta. Lower border of sternum. Murmur late (S.M.).

THE TRAINING OF STUDENTS IN GENERAL PRACTICE

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The demands of general practice are too wide to enable medical students to be trained during their undergraduate curriculum in all the experiences they are likely to meet as general practitioners.

Medical education must include in its objectives more than the mere teaching of facts and techniques. It must prepare the student adequately and intelligently to meet situations he has never met before, whether concerning individuals, families or communities. He must learn to shoulder responsibility and be self-reliant, to observe and make inferences from his observations and to learn and practice the principles of methodology. His training from his first to his final year should foster a desire to understand the rationale of human reactions and behaviour; he must learn how to know people, their illnesses, and their neighbours' illnesses. He must know what is normal in individuals and communities, both physically and psychologically, and he must know how

to maintain that state of normality. Most of these qualities do not belong to any particular field of work or to any particular subject in the curriculum; they can and must be developed as a philosophical theme in all subjects.

Nevertheless, there are basic facts and experiences that must be taught and learned in every subject, in the 'subject' of general practice no less than any other. How can students be taught the experiences or some of the experiences of general practice, and at the same time learn to appreciate and apply the above-mentioned general philosophical principles? Attempts are being made to provide the practical answer to this question in many medical schools of the Western world.

Common to all these attempts is the providing of clinical experience for the students outside the wards and outpatient departments of the teaching general hospital. It is claimed that this kind of practice provides certain types of experience that cannot be obtained in a general hospital. These types of experience include the management of minor illness that is not serious enough to be admitted to hospital, the observation of normal people, the management of emergencies in an environment devoid of the facilities that are available in a hospital, of old people, and of chronic and incurable illness in the home environment; the kindly but rapid disposal of trivial complaints and the differentiation of these from *apparantly* trivial complaints which are indicative of serious physical or mental disease; the observation of the natural course of disease; the appreciation of medico-legal facets to cases and the applications of the law in practice, for example, in public health matters and in the certification of mental disorders; the use and value of social-service agencies such as those concerned with child welfare, or the care and rehabilitation of alcoholics; and the impact of illness, both physical and mental, on persons other than the patient. In the general-practice training schemes to be described, this experience is obtained in an extra-hospital environment under the guidance and supervision of practitioners experienced in general practice.

Clearly, some of these experiences occur in the wards and out-patient departments of general hospitals and in the ordinary social contacts of everyday life, but these self-same experiences carry a different educational value when experienced outside the hospital environment.

The schemes adopted in numbers of medical schools to provide 'general-practice experience', some of which schemes I visited personally in 1953, have been classified by the British Medical Students' Association in their published survey of schemes in operation in British medical schools which they presented to delegates of the first World Conference on Medical Education in London in August 1953. This survey classifies the schemes as follows:

1. *Health-Centre schemes.* The Edinburgh scheme, which I visited, will be described, and also the proposed scheme at Manchester.

2. '*Residential*' *schemes*, where students spend a period of 2—4 weeks living with a general practitioner, attending his 'surgery' hours and accompanying him on his rounds. I discussed this type of scheme at Aberdeen, where it is voluntary, and at Charing Cross, London, where it is compulsory.

3. '*Attachment*' *schemes*, where the student is attached to a general practitioner for the mornings only, for a period usually of about 2 weeks.

4. '*Day visit*' *schemes*, where the student spends an occasional day with a general practitioner.

In the United States, schemes corresponding to the residential and the attachment schemes are under trial, as well as schemes not very dissimilar from the British health-centre schemes or that at the University of the Witwatersrand.

In Norway, training in the experiences of general practice will shortly be made obligatory after graduation; 6 months of the year's compulsory internship will be spent as assistant to the equivalent of the part-time District Surgeon in South Africa.

The scheme at the Alexandra Health Centre and University Clinic (Witwatersrand University) approaches most nearly the British health-centre scheme of all overseas schemes studied, yet with quite considerable differences both in the type of experience offered and in the general philosophical principles learned by the students.

I shall briefly describe and comment on certain representative schemes at Aberdeen, Edinburgh, Charing Cross and Manchester, where I visited and the scheme at Richmond, Virginia, described at the Conference on Medical Education; and also the Witwatersrand scheme.

Aberdeen. During medical-school vacations, each of 15—20 general practitioners in Aberdeen and the countryside around is allocated one senior student for a month. The student does not as a rule reside with the practitioner, but accompanies him in all his work, both office and domiciliary. The scheme is voluntary, both for the students and the practitioners, and about two-thirds of the students volunteer for this experience. When, some years ago, the Medical Faculty at Aberdeen approached the local branch of the British Medical Association for their views on instituting a general-practice training scheme, the branch did not accept the scheme because they feared that the patients would object. A year or two later, the branch reconsidered their refusal, and asked the University to give the scheme a trial on a voluntary basis. This is now being done, and the present scheme is regarded as a success. The students like it, the practitioners acknowledge that the students keep them up to the mark and on their mettle, and the patients are intrigued by this new interest which is being taken in their health. The students gain experience in general practice, but do not undertake much responsibility. They do not learn to know people as intimately as they would if they were given more responsibility.

Edinburgh. The General-Practice Training Unit at Edinburgh has been established for the specific purpose of training students. The Unit functions as a general-practitioner unit under the National Health Service, and is in two sections, one at the Royal Dispensary in West Richmond Street, and one at the Livingstone Dispensary in the Grass Market. Each of these sections

has a 'panel' of 2,000 patients and potential patients, which is regarded as an average-sized practice for one general practitioner in the National Health Service. As it is a teaching unit, two practitioners (instead of one) are attached to each section of the unit. At the Royal Dispensary, there is also a social worker, a nurse and a secretary. Regular 'surgery' hours are held twice a day, and domiciliary visits are paid. The patients attending offer a cross section of general practice in a poor district of Edinburgh.

Students' attendance is not full-time; a group of 6 attend part-time over a period of 10 weeks. Each student sits with one of the doctors at a surgery session twice a week. From the contacts made at these 'surgery' sessions arises the rest of the student's work during his period in the unit. The student attends to the case notes, examines the patient, and is made to feel that he is one of the team. His follow-up of the cases includes domiciliary visits, alone, or with a doctor if the likely management of the case is beyond his experience, and he accompanies the patient to the Royal Infirmary for consultations, or for special investigations such as X-rays. He 'sees the patient through' the various departments of the Royal Infirmary on such occasions. He becomes closely acquainted with the patient and with the patient's relatives in this way, and learns to appreciate the personal implications of the procedure of 'investigating' a case. He meets the social worker and the pharmacist over individual cases at the Dispensary, and the whole attending group of students attends a conference once a week with the medical, nursing and social-worker staff. Only one-quarter of the total number of Edinburgh medical students at present passes through this Unit during the medical curriculum, owing to the large number of students still being admitted to the medical school.

The cost of the Unit is about £19,000 per annum, which is being found by the University, the National Health Service and the Rockefeller Foundation.

An alternate scheme of reducing the period of 10 weeks in this Unit to 6 weeks has been discussed, the students for the remaining 4 weeks to be 'apprenticed' to general practitioners. This would enable more students to pass through the General-Practice Training Unit in their medical course.

In this 'Health Centre' form of training for general practice, students learn responsibility in that they are made partially responsible at least for patient welfare and therapy. They act under the guidance of experienced practitioners, but perhaps see too comfortable a form of general practice. To a limited extent they learn to decide issues of patient management themselves. They see the impact of illness in the home environment, and upon earning capacity. The seeds of social conscience are permitted to develop.

Manchester. 'Derbyshire House', the General-Practice Training Unit of Manchester, was not yet functioning as such at the time of my visit. It is intended that the Unit shall function in much the same way as the Unit at Edinburgh, but with a different method of staffing. At Edinburgh all the medical staff are full-time and are paid by the University. At Manchester it is proposed that there shall be a full-time Director, and in addition 4 local general practitioners employed part-time. At Derbyshire House, which is situated not far from the Manchester Royal Infirmary, the University will provide 4 suites of consulting rooms, each suite being a complete 'surgery' for the use of one general practitioner. The students will attend the practice in the surgery and also accompany the general practitioner on domiciliary visits. The general practitioners will be selected from the local practitioners of Manchester, and will continue in their own general practices. The time to be spent in the Unit by the students is still to be decided.

Charing Cross. Charing Cross Medical School, one of the smaller and rising London schools, is one of the few that has instituted a compulsory period of attachment of students to general practitioners. Students at some time after passing their pathology examination, which is written 6 months before the final M.B. examination, are each allocated full-time to a local general practitioner for a period of 3 weeks. They attend with the general practitioner to all aspects of the work of that particular general practice. The Medical Faculty invites general practitioners to join their panel of teachers, and from these volunteers selects teachers on personal and geographic grounds.

Richmond, Virginia. The scheme in force at Richmond is organized on a rather more elaborate scale than corresponding schemes in Great Britian. Four medical officers trained in general practice have been added to the teaching staff of the medical school, with a secretary and a telephone exchange operator. Six or 7 students at a time are attached for training to the Unit. They make domiciliary visits in motor cars equipped with two-way radio telephones and, if in difficulties, they are able to communicate directly with their seniors at headquarters. It would appear that one at least of the objectives in the training of medical students in general-practice experience, namely, the undertaking of responsibility, is apt to be missing in such an elaborate scheme as this.

Alexandra Health Centre and University Clinic. The training scheme of the Witwatersrand University at the Alexandra Health Centre and University Clinic is a 'Health Centre' type of general-practice training unit. The students in groups of 5 or 6 in their final year live in at the Clinic for 2½ weeks. A vast out-patient clinic and a limited domiciliary service is provided for the African population of Alexandra Township. The large attendance of patients at the Clinic necessitates the provision of a rapid service, the students taking an active part in providing this service, which includes attending to sick and injured adults and children, pregnant mothers, and patients suffering from chronic contagious diseases such as tuberculosis. The students undertake responsibility, but when so doing have the 4 medical officers of the staff to call upon for guidance, and a number of part-time visiting staff. They pay a certain number of domiciliary visits in the Township, either in the Clinic cars or on push bicycles, the riding of which is a feat on the unmade roads of the Township.

The cost of the Clinic is £25,000 a year. It provides primarily a service to the local population and, in passing, a very fine teaching service for medical students. The students see the living conditions of the lower socio-economic groups at their worst, and any seeds of social service are nurtured by the 2½ weeks' experience.

DISCUSSION

Common to all these schemes is the experience of seeing patients outside the general hospital in the more natural environment of the home and the community. Short as the period of such experience may be, it is sufficient to enable the student to picture his hospital patients in the environment to which they return on leaving hospital.

In schemes like those at Edinburgh and at the Alexandra Township students learn self-reliance and responsibility, in that they have to decide whether to ask for guidance in the management of patient situations or cope with such situations themselves.

They experience for the first time the traditional confidence which is placed by patients in the doctor. Some preen themselves in this reflected glory of an age-old profession, some pride themselves and some are humble and rather frightened by the trust and confidence that is placed in them. It is revealing to see that on occasions the patients place more confidence in their medical-student 'doctor' than in the consultant who happens to be called in to see their case.

The social conscience, even if present in the most embryonic form, is stimulated by experience of this type, particularly where the environment is as dramatically sub-economic as that of Alexandra Township.

The system of apprenticeship to general practitioners enables the student to see patients in their home environment, and to see a cross section of the work of the general practitioner. It lacks the facilities that permit the student to undertake responsibility, and to get to know people. From the general practitioner's point of view, the effect is the same as that which the student has upon the hospital teacher, putting him on his mettle and stimulating constant attention to his patients.

There are difficulties in the administration of the apprenticeship system. Personal incompatibilities between student and practitioner, which are bound to occur, have a greater impact on both student and teacher than in a hospital teaching scheme where the multiplicity of teachers smooths over the effects of individual incompatibilities. The geographic and multi-racial aspects of practice in South Africa add difficulties probably peculiar to South Africa.

By and large, the apprenticeship system would appear to be less effective and constructive than the health centre system.

'Attachment schemes' and 'day visit schemes' have very little to commend them as part of the medical curriculum. Voluntary attachments of this type during vacations may have an interest-stimulating effect.

In deciding whether to introduce a compulsory training scheme of one of the types described, a medical faculty should give due weight to the opinion held by many authorities that an adequately accommodated and staffed hospital out-patient department, with a social workers' service included, and with facilities for domiciliary visiting by students, provides experience equivalent or superior to any of the general-practice training schemes described.

The best way to provide experience equivalent to general practice or in general practice itself will depend on the local conditions that apply at each medical school.

BISMUTH EDTA COMPLEX

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The water-soluble stable chelated compound, lead ethylenediamine tetraacetic acid (lead EDTA complex), has been shown experimentally to have value as a contrast medium for oral and parenteral administration.[1, 2, 3] The less soluble lead calcium EDTA complex also produces good shadows of the alimentary canal, but by intravenous injection it does not produce shadows of the kidneys.[4]

It was considered desirable to investigate other metallic complexes of related type for possible radiographic use. Since certain bismuth compounds have been used in diagnostic radiology, it was of interest to study the bismuth complex of ethylenediamine tetraacetic acid. The atomic weight of bismuth is 209, as compared with that of lead, which is 207. The bismuth compounds used in the past proved toxic and have been superseded by other radiopaque compounds. Since the complexes formed by ethylenediamine tetraacetic acid with divalent and trivalent metals are stated to be generally stable, soluble, unhydrolysed compounds, in which the metal is bound in a form which is inactive, it was considered that this type of chelated bismuth compound might prove of more value. A supply of the bismuth complex was prepared by the Geigy Company, and some of its properties are reported here.

Experimental Results

Bismuth EDTA complex proved to be less soluble than the lead complexes previously investigated. The highest concentration prepared in warm Locke's solution was a 2.5% solution, and this was used throughout the investigation. The pH value of this solution was 2.18.

Radiography. The solution was administered orally through a stomach tube to adult albino rats, which were subsequently anaesthetized for radiography. A shadow of the gastro-intestinal tract was obtained, not of very

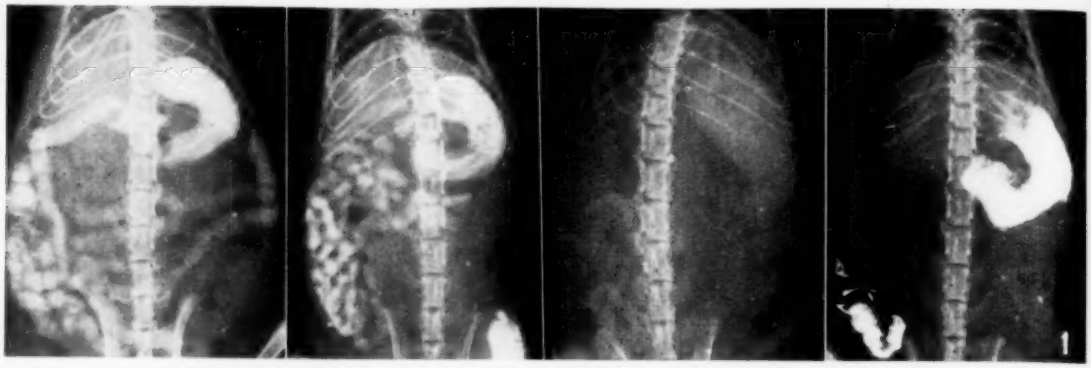

Fig. 1. Radiographs of alimentary canal (rat) to show relative densities of solutions of contrast media 1 hour after administration. Left to rght: lead EDTA 25% (1 ml.); lead calcium EDTA 15 % (1 ml.); bismuth EDTA 2·5% (2 ml.); emulsion of barium sulphate 1 ml.

dense degree and provided 2 ml. or more of the solution was administered (Fig. 1).

The intravenous injection of 2 ml. of the solution failed to produce shadows of the renal calyces and pelves in rats.

Toxicity. No ill effects were observed clinically in rats to which 2 ml. doses were administered orally. Rats which received 1—2 ml. of the solution intravenously during anaesthesia for urographic studies made good recovery. Intraperitoneal injection of large doses (5 ml.) produced death within 12 hours of administration.

Intravenous injection of 0.25—1 ml. per kg. bodyweight in anaesthetized cats produced depression of auricular and ventricular contractions and disturbances of conduction of longer or shorter duration, depending on the dose administered; there was some decrease in the blood pressure and marked fluctuations at the lower and the higher levels until the return to normal. The effects recurred with repeated doses. Atropine did not prevent the effects. Recovery ultimately occurred from each dose. The heart was not arrested. The changes, due to a direct action of the complex on the myocardium, were demonstrated on kymographic and electrocardiographic tracings (Figs, 2 and 3.). The latter showed bizarre changes in the complexes. Intestinal volume recording revealed no significant change in the size of the blood vessels.

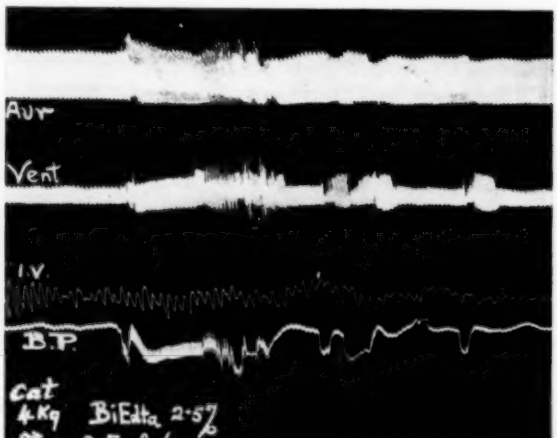

Fig. 2. Cat 4 kg. Effect of bismuth EDTA 2·5% (0·5 ml. per kg.) on auricular and ventricular contractions, intestinal volume, and blood pressure.

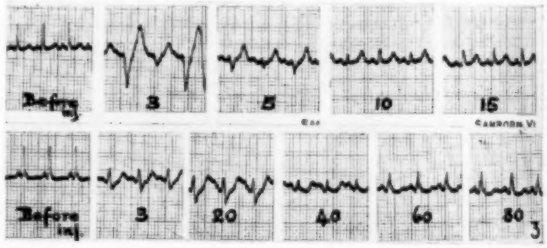

Fig. 3. Electrocardiographic changes produced in cats by bismuth EDTA complex 2·5% solution.
Upper tracing: before injection and 3, 5, 10, 15 minutes after injection of 0·25 ml. per kg.
Lower tracing: before injection and 3, 20, 40, 60, 80 minutes after injection of 1·0 ml. per kg.

The contractions of isolated segments of the small intestine of the cat are not affected even by high concentrations (1 in 2,000).

Discussion

Oral administration of a solution of 2.5% bismuth EDTA complex produces a shadow of the stomach and intestine much less dense than that produced by the more soluble lead complexes. A greater density of shadow would be obtained if a suspension of the complex were used. Before barium sulphate replaced insoluble bismuth salts as a diagnostic aid in gastro-enterology, bismuth subcarbonate was administered in suspension in doses of 50 g.

Intravenous injection of the bismuth complex was

unsatisfactory for urography. This is again due to the relatively low solubility of the compound and the low concentration and dose that can be injected. Lead calcium EDTA also failed in this respect presumably for the same reason. On the other hand the very soluble lead sodium EDTA proved satisfactory for intravenous urography. The bismuth complex produced marked changes in the heart action, but recovery occurred even when the kymographic and electrocardiographic records suggested profound changes in myocardial activity. The myocardial changes may be due to altered (intracellular) electrolyte ratio as suggested for lead EDTA complex.[2, 3] They are presumably not due to the action of bismuth, which is apparently firmly chelated and unionized in the EDTA complex. It is interesting for comparison that intravenous injection of the usual soluble bismuth preparations leads to dangerous and even fatal (flocculation) shock due to the formation of insoluble compounds. The fall of blood pressure produced by such soluble bismuth salts generally recovers promptly; it is chiefly cardiac, due to disturbed conduction and heart block.[5] The kidneys, liver and other organs may also be affected by these soluble bismuth salts. Such effects have not been studied as far as bismuth EDTA complex is concerned.

SUMMARY

Bismuth EDTA complex in 2.5% solution gives a shadow of the stomach and intestines. A larger dose must be administered than with the more soluble lead complexes and the shadow is not so dense. By intravenous injection it is not satisfactory for urography. It produces depression of the heart and striking electrocardiographic changes, from which recovery occurs.

The bismuth complex was supplied as 'Sequestrol-bismuth complex NA' by the Geigy Company Ltd., Rhodes, Middleton, Manchester, to whom grateful acknowledgment is made. Expenses were partly defrayed by a grant from the C. L. Herman Research Fund. Mr. J. W. Bates assisted in the investigation. Dr. B. Isaacson recorded the electrocardiograms.

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IN PARLIAMENT

THE REVIVED SUPPLEMENTARY HEALTH SERVICES BILL

From a Parliamentary Correspondent

Among the Bills introduced at the outset of the current parliamentary session, the one of most direct interest to the medical profession is the revived Supplementary Health Services Bill, whose purpose is 'to provide for the registration and control of the activities of persons engaged in the performance of supplementary health services . . . .' For all practical purposes it is identical with the measure that was introduced in 1952 but was allowed to lapse in the following year.

This one is intended to be piloted in due course by the Minister of Health, Mr. J. F. Naudé. In view of its important bearing on the livelihood of many persons and the wide interest and conflicting views to which it gave rise at its first publication the Bill has been referred to a Select Committee.

The classes of persons to whom it is contemplated the Act will apply are listed in a schedule to the Bill as follows: chiropodists, diagnostic radiographers, dietitians, food inspectors, health inspectors, hygiene officers, masseurs, medical technologists, occupational therapists, optometrists, orthopaedic mechanicians and surgical appliance makers, orthoptists, physiotherapists, radiographers and speech therapists.

However, the Bill provides in clause 2 (2) and (3) that the Governor-General may at any time, upon the recommendation of the South African Medical and Dental Council, by proclamation in the *Government Gazette* amend the schedule by including any class of persons engaged in the performance of supplementary health services; and that the Minister of Health may, whenever he deems it necessary, by notice in the *Gazette*, define the scope of the calling of any class of persons engaged in the performance of supplementary health services.

The Medical and Dental Council will maintain registers for persons engaged in the various classes of supplementary health services. It may hold, or arrange for, examinations for any class for whom a register is required to be kept, and it may prescribe the certificates of competency, granted after examination, which will entitle anyone to registration in the relevant class. If the Council prescribes a certificate issued by an examining authority outside the Union it must be satisfied that the standard of training indicated is not lower than that represented by a certificate granted by the Council and entitling the holder to be registered.

LIMITED SELF-ADMINISTRATION

Each class will have a form of limited self-administration through a committee. The Medical and Dental Council may make rules within the framework of the intended Act, provided it consults the committee or committees concerned and obtains the approval of the Minister of Health. These rules may prescribe the standard of general education required of candidates entering for examination for certificates of competency, the persons who may be admitted to such examinations, the courses of study and the training required and other such details.

The rules may also prescribe the conditions under which any class of persons to whom the Act applies may carry on their callings, the degrees, diplomas and certificates that may be registered under the Act, and the titles which may be used by any class of persons registered. The Bill is designed to take effect piecemeal for the various classes, the dates being fixed by proclamation in the *Gazette*. Twelve months' grace will then be allowed, but after that any unregistered person performing for gain any act pertaining to the calling of the relevant class or holding himself out to be entitled to perform any such act will be liable to a fine not exceeding £50.

Any person registered under the Medical, Dental and Pharmacy Act when this Bill takes effect will be deemed to be registered in terms of the new measure.

The committee for any class covered by the Bill will include one or two persons appointed by the Medical and Dental Council from among its members, but the Bill is so designed that committee members elected within the class will be in the majority. All the committee members will have to be European South African citizens.

Section 20 of the Bill ensures that no individual should become involved in any conflict between its terms and those of the Medical, Dental and Pharmacy Act. Once a class of persons to whom any provision of the Medical Act applies is proclaimed to be subject to the terms of the Supplementary Health Services Act, then the relevant provision in the Medical Act will cease to affect that class.

NURSING AMENDMENT BILL

The 1955 version of the Nursing Amendment Bill, which has also been published, increases the medical profession's influence on the South African Nursing Council. Whereas before the Minister of Health was to have appointed to it two persons who were not registered in terms of the Medical, Dental and Pharmacy Act or the Nursing Act, he will now appoint in addition one person registered under each of these Acts, respectively. The one registered in terms of the Medical Act will be required to have special knowledge of, or experience in, obstetrics.

In addition the Medical Council will be required to appoint a dentist, as well as the two medical practitioners specified in the 1952 Bill. All the members of this Council, too, will have to be European South African citizens. The Council will have to exercise surveillance over the conditions in which nurses and midwives train. As an ultimate sanction it would be able to withdraw its approval from any training school.

FRIENDLY SOCIETIES BILL

Another measure that has been resuscitated and is now in the hands of a Select Committee, is the Friendly Societies Bill. Its oblique interest to the medical profession lies in the fact that a system of registration and supervision is being applied to institutions which include in their objects 'the provision of or the payment of contributions towards the cost of medical, nursing, surgical, optical or dental attendance or medicines or other medical requirements . . . .'

POLIOMYELITIS IN THE UNION

Following are the returns, supplied by the Union Department of Health, of cases notified under the Public Health Act as suffering from Poliomyelitis in the period 14 to 19 January 1955.

	European	*Non-European*
Transvaal:		
Johannesburg	7	1
Pretoria	5	
Pilgrimsrest district		1
Middelburg district		1
Middelburg Municipality	1	
Brakpan	1	
Springs	1	
Louis Trichardt	1	
Waterval Boven	1	
Vereeniging P.U.A.H.B.		1
Lyttleton	1	
Standerton	1	
Roodepoort-Maraisburg	1	
Total for Transvaal	20	4
Cape Province:		
Cradock Divisional Council		2
Matatiele Municipality	2	
Matatiele district		1
Molteno		1
Indwe Divisional Council		1
East London Municipality	1	
Umzimkulu district		2
Port Elizabeth	1	
Total for Cape Province	4	7
Natal:		
Durban	2	
Pietermaritzburg	2	
Pietermaritzburg district		1
Marianhill		1
Pinetown district		1
Umzinto ,,		1
Empangeni		1
Inanda district		1
Camperdown		1
Stanger		1
Camperdown district		1
Ladysmith	1	
Malvern	1	
Ndwedwe		1
Total for Natal	6	10
Orange Free State:		
Parys	1	
Kestell	1	
Rouxville district	1	
Marquard district	1	(died)
Frankfort district		1
Vierfontein (Bothaville district)	1	
Ventersburg	1	
Total for O.F.S.	6	1
TOTAL FOR UNION	36	22

Union Department of Health Bulletin. Report for the 7 days ended 20 January 1955:

Plague, Smallpox: Nil.

Typhus Fever. Orange Free State: Four (4) Native cases and one (1) Native death in the Parys district. Diagnosis based on clinical grounds only. *Cape Province:* No further cases have been reported from the Aliwal North and Lusikisiki districts since the notifications of 22 December 1954. These areas are now regarded as free from infection.

Epidemic Diseases in other Countries:

Plague: Nil.

Cholera in Dacca (Pakistan); Calcutta, Madras (India).

Smallpox in Lahore (Pakistan); Basra (Iraq); Bombay, Calcutta, Delhi, Jodphur, Kanpur, Madras (India); Moulmein (Burma); Phnom-Penh (Cambodia); Saigon-Cholon (Viet-Nam).

Typhus Fever: Nil.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Maybaker (S.A.) (Pty.), Ltd., announce the introduction of *Hydroxystilbamidine isethionate.* This compound is similar in activity to stilbamidine but does not cause trigeminal neuropathy and is more stable in aqueous solution.

It is as effective as stilbamidine in allaying the pain of multiple myeloma and sometimes temporarily arresting the malignant process, and is to be preferred in the treatment of this condition on account of its freedom from toxicity.

Hydroxystilbamidine has given promising results in such increasingly common systemic fungal infections as those due to *Aspergillus* and *Candida* species. The compound has been found to be curative in blastomycosis due to *Blastomyces dermatitidis* and in coccidioidomycosis. Its trial is also suggested in infections due to *Cryptococcus* species. This drug is also as effective as stilbamidine in both fresh and relapsed cases of Kala-azar.

Hydroxystilbamidine is usually given by slow intravenous injection. The intramuscular route is not recommended.

Available in cartons of 10 × 250 mgm. ampoules of the pure substance.

J. H. G. Geer
Technical Information Department

LEPROSY CONFERENCE, PRETORIA, 1954

A 3-day Conference on Leprosy was held on 19-21 October at Westfort Institution, Pretoria, on the recommendation of the South African Leprosy Advisory Committee. Representatives were present from the South African Leper Institutions, the Protectorates, neighbouring territories and the Advisory Committee. The Conference was opened by the Secretary for Health, Dr. J. J. du P. le Roux, who gave a review of the history of Leprosy in South Africa and showed how, due to the aid of modern treatments, we could now claim that leprosy was under control in the Union.

Papers were read on the classification and histopathology of leprosy (the Union has accepted the classification laid down at the 1953 Madrid Congress). Erythema Nodosum Leprosum, which is a frequent complication of leprosy, was dealt with from its clinical and histological aspects. It was shown to be a panniculitis—in fact a case of panniculitis was recorded in the literature in which only the occurrence of a second case revealed that the primary disease of the pateint was leprosy.

The sulphones are the basis of the modern treatment of leprosy; papers were presented showing the benefit, or otherwise, of thiosemicarbozone, isonicotinic acid hydrazide, ACTH and atabrine as additive treatments. The significance and treatment of positive serological tests for syphilis was dealt with, as also were the lepromin tests, the tuberculin test, and B.C.G. in leprosy. Details were given of operations for leprotic neuritis and two papers on blood chemistry and tongue patterns were presented.

The effect of sunlight on the staining properties of the leprosy bacillus, and the significance of the finding of scanty bacilli, were detailed in two papers. 'Primary pigmentation of the skin caused by long-wave ultra-violet rays of sunlight in South Africa' was the title of another paper.

Pictures of the leprosy bacillus taken by the electron microscope were demonstrated. An investigation was detailed on the question whether compulsory segregation led to the hiding of cases. Details were given of staff members who have become infected in South Africa and other parts of the world.

A copy of the proceedings may be obtained from the Medical Superintendent, Westfort Institution, Pretoria.

Titles of Papers: A. R. Davison: 1. Classification of Leprosy, 2. Erythema Nodosum Leprosum, 3. Treatment by Thiosemicarbozone, 4. Treatment by ACTH, 5. Treatment by Atebrine, 6. Treatment by Isonicotinic Acid Hydrazide, 7. Significance of 'Very Scanty Positive Smears', 8. Does Compulsory Segregation lead to hiding of Cases of Leprosy? 9. Infections in Institutions.

R. Kooij: 1. Histopathology of the Different Types and Groups of Leprosy, 2. Histopathology of Acute Panniculitis Nodosa Leprosa (Erythema Nodosum Leprosum), 3. Effect of Sunlight on the Staining Properties of the Leprosy Bacillus, 4. Lepromin Test, Tuberculin Test and BCG in Leprosy, 5. Staining of Leprosy Bacilli by the Ziehl Neelson Method, 6. Primary Pigmentation of the skin by the Long Wave Ultra-violet Rays of the Sunlight in South Africa, 7. Study by Electron Microscopy of the Morthology of Mycobacteria Leprosy.

H. Mostert: 1. The Classification of Leprosy.

H. F. Schiller: 1. Interpretation of the Positive Serological Test for Syphilis and the Management of Latent Syphilis.

I. le Roux: 1. Nerve Operations.

A. A. Kinnear: 1. Chemistry of the Blood in Leprosy.

B. T. Squires: 1. Human Tongue Patterns.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Sam Miller, M.R.C.P., D.G.O., D.C.H., M.M.S.A., of Port Elizabeth, has recently returned after 2 years' post-graduate study and one year's teaching in Gynaecology and Obstetrics at the Rotunda Hospital, Dublin.

BOOK REVIEWS : BOEKRESENSIES

SEX IN MARRIAGE

Sexual Adjustment in Marriage. By Henry Olsen, M.D. (Pp. 310 + xxxiv. 21s.) London: George Allen & Unwin. 1954.

Contents: 1. The Human Organism. 2. The Sex Organs. 3. Hormones and the Sexual Functions. 4. Sexual Need or Sex Urge. 5. Living Together. 6. Hygiene of the Couple's Sex Life. 7. Disturbances in a Couple's Sex Life. 8. Marriage. 9. Sex Life of the Unmarried. 10. Fertilization, Pregnancy, Childbirth. 11. Interruption of Pregnancy. 12. Regulation of Fertilization. 13. Masturbation. 14. Sexual Abnormalities. 15. Diseases of Sex Life. 16. Sexual Education.

This book has been written for the instruction of the lay public. The author is a medical man, and he has carried out the task he set himself with delicacy and a deep sincerity. The performance of this duty was perhaps not so onerous to the author as it would be to most of us, for at an early stage of his career Dr. Olsen was appointed by the City of Copenhagen as organizer and director of instruction in sexual hygiene to certain students for whom the Copenhagen Municipality is responsible. Sexual instruction has since been adopted by most Danish colleges and in 1945-46 it was introduced in all Copenhagen public schools and high schools and in many corresponding schools all over the country. Dr. Olsen's *Textbook on Sexual Hygiene* is widely used in these schools.

The present work consists of 16 sections which cover 67 chapters and 667 numbered items. There is no further index. The subject matter dealt with is in effect an account of the biology of reproduction. Against this general background there is brought into relief the problem of sexual adjustment in marriage. After a general biological consideration anatomy is presented, then physiology, sometimes some pathology and, finally, a certain emphasis on the psychological and psychiatrical. This is rounded off with a substantial account of venereal disease.

The author never fails to show good taste. The only gross inaccuracy discovered was the statement that the urethra was 2½ to just over 3 inches in length, when presumably centimetres appeared in the original language. The information and advice given to readers is acceptable, and here the author proves himself to be a highly responsible person. The author himself clearly exhibits purity of heart and he labours under a desire to raise marital success to a high level, being convinced that a fuller understanding of the biological and psychological relevancies would at present promote such an endeavour.

O.S.H.

METABOLIC AND TOXIC DISEASES OF THE NERVOUS SYSTEM

Metabolic and Toxic Diseases of the Nervous System. Proceedings of the Association for Research in Nervous and Mental Disease. Edited by H. Houston Merritt, M.D. and Clarence C. Hare, M.D. (Pp. 604 + xii, with 104 illustrations and 73 tables. 76s. 6d.) London: Baillière, Tindall & Cox. 1953.

Contents: 1. The Parathyroid Gland and its Relationship to Diseases of the Nervous System. 2. The Pituitary Gland in Relation to Cerebral Metabolism and Metabolic Disorders of the Nervous System. 3. Studies of Adrenocortical Physiology in Relation to the Nervous System. 4. The Thyroid Gland in Relation to Neuromuscular Disease. 5. The Thymus and its Relationship to Diseases of the Nervous System: Study of 374 Cases of Myasthenia Gravis and Comparison of 87 Patients Undergoing Thymectomy with 225 Controls. 6. Clinical and Physiological Effects of Excessive Heat. 7. Clinical and Metabolic Effects of Exposure to Low Environmental Temperatures on the Central Nervous System and Related Visceral Structures: A Cytochemical Study. 8. The Role of B Vitamins in the Metabolism of the Nervous System. 9. Folic Acid, Vitamin B^{12}

and Related Compounds in Metabolism of the Nervous System. 10. Abnormal Copper Metabolism and Hepatolenticular Degeneration. 11. The Neurological Disorder Associated with Liver Disease. 12. Diseases of the Nervous System Associated with Disturbances of Lipide Metabolism. 13. Neurophysiological Aspects of the Opiate and Barbiturate Abstinence Syndromes. 14. Psychoses Produced by Administration of Drugs. 15. Physiologic Development of the Cortex of the Brain and its Relationship to its Morphology, Chemical Constitution, and Enzyme Systems. 16. A Quantitative Histochemical Study of Eight Enzymes of the Cerebellar Cortex and Subjacent White Matter in the Monkey. 17. Carbohydrate Metabolism and Diseases of the Nervous System. 18. Hypoglycemia and Brain Metabolism. 19. Potassium Metabolism and Dysfunction of the Nervous System Associated with Hyper- and Hypokaliemia. 20. Porphyrin Metabolism and Diseases of the Nervous System. 21. Creatine and Creatinine Metabolism and Diseases of the Neuro-Muscular System. 22. Biochemical Actions of Toxic Substances on the Nervous System. 23. The Toxic and Metabolic Effects of General Anaesthetic Agents on the Central Nervous System. 24. Effects of Ionizing Radiation on the Adult and Embryonic Nervous System. 25. Effects of Heavy Metals on the Central Nervous System. 26. The Effects of the Organic Solvents and Industrial Poisonings on the Central Nervous System. 27. The Effects of Bacterial Toxins on the Nervous System. 28. The Effect of Alcohol on the Nervous System. List of members. Index.

The Proceedings of the Association for Research in Nervous and Mental diseases are justifiably regarded as an up-to-date epitome of research and an authoritative review of modern ideas, and are an indication of the very high standard that has been attained in neurology and in psychiatry in the western world. It has been the custom to deal with a specific field or subject each year so that each volume in the past has not only contained papers of a high standard, but has been sufficiently comprehensive in its selection to justify its being used as a book of reference. The present issue, however, attempts to cover an area so wide that completeness appears, to some extent at any rate, to have been sacrificed for variety and this reviewer was left at times with a sense of omission, and at other periods with a vague feeling of dissatisfaction and even of irrelevancy.

Each of the 28 chapters well maintains the expectedly high standard. The opening paper on the parathyroid is one of the best in the book, and is followed by a series of articles on endocrines; but, inexplicably, hypothyroidism as a factor in syndromes of the nervous system receives no mention at all. The inclusion of the chapters on the clinical effects of excessive heat, and of cold, come almost as an intrusion! Both, however, are informative and represent original work; the section dealing with the effects of cold should be read by every neurologist; it is interesting to note that of 112 references, only 2 report on the infamous work of the Nazis.

Vitamin-deficiency syndrome are very inadequately represented, but the reports on copper metabolism and hepato-lenticular degeneration, and the stress laid on liver disease in the etiology of neurological syndromes and coma are well worthy of study; the relationship between hepatic pathology and brain disorders will perhaps be increasingly recognized in the future. A well documented article on hypoglycaemia still leaves us in the dark as to how insulin therapy produces its benefits, or whether it produces any at all! Finally there are educative chapters on the effects of drugs, organic solvents, anaesthetics and such like toxins.

Like its predecessors, this issue is a 'must' in the library not only of every neurologist or psychiatrist, but of anyone interested in metabolic disorders. Most of the clinical papers deal with both neurological and psychiatric aspects, thereby demonstrating the close relationship between the two. Finally, the discussions at the end of each paper are themselves very informative, though it is a pity they are so condensed.

M.P.

CONGENITAL SYPHILIS

Congenital Syphilis. By David Nabaroo, M.D., F.R.C.P. (Pp. 470 + xi, with 95 illustrations. 50s.) London: Edward Arnold (Publishers) Limited. 1954.

Contents: 1. Nomenclature. 2. Historical. 3. Incidence of Congenital Syphilis. 4. Transmission of Congenital Syphilis. 5. Pathology. 6. The Clinical Picture. 7. Symptomatology. 8. Symptomatology (continued). 9. Third-Generation Syphilis. 10. Familial Susceptibility of Individual Tissues to Syphilitic Invasion. 11. Diagnosis and Prognosis of Congenital Syphilis. 12. Treatment of Congenital Syphilis. 13. Acquired Syphilis in Childhood. 14. Syphiloids. Index to Authors. Subject Index.

This is a new book by a paediatrician who has shown particular interest in congenital syphilis for the greater part of his clinical years. His observations are, therefore, of special interest.

He notes that syphilis and tuberculosis are not infrequently associated in affections of the lungs, and reports on 20 cases personally observed. The literature reports few such cases in children. The author considers that paroxysmal haemoglobinuria is often caused by syphilis because he finds the condition clears up on treatment with anti-syphilitic drugs. He advises that every case of acute or chronic lymph-node enlargement should be suspected of being syphilis, and that biopsy of the glands and serologic tests should be carried out. Mild hydrocephalus, herpes zoster and convulsions are not infrequently associated with congenital syphilis. Congenital syphilitics may have psychical manifestations as their only signs or symptoms. Thus delinquents, mental deficients, and cases of chorea, should be investigated for syphilis, as should be their parents and other members of the family. A notable omission from among the causes of false positive blood-reactions is glandular fever (infectious mononucleosis).

A feature of the book is the excellent case histories from the author's own material. The length of time over which some of his cases have been observed can be judged when it is seen that many of them were originally treated with mercury, the majority with arsphenamine and bismuth; all belong to the pre-penicillin era. The treatment advised by the author is penicillin, mapharside and bismuth, given during the same week. This differs from the American method of using penicillin only.

Nabarro considers third-generation syphilis possible. He gives details of 8 families with syphilis in 3 generations. He believes that a woman with untreated congenital syphilis may have children whose developing tissues and organs are adversely affected by the maternal syphilis without there being active syphilis in the infant. Anti-syphilitic treatment of such infants would be of no avail. He names this condition 'para-syphilis' or 'occult syphilis.' Both these names have been given to entirely different conditions by other writers.

The author notes that in Britain there is a real reduction in the prevalence of congenital syphilis. This has resulted in the teaching that the disease hardly exists and need no longer be considered. He points out that there is a real danger in this state of complacency. It is of interest to note that the cutting down of the Federal Government vote for the treatment of syphilis in the United States of America resulted in an increase of syphilis in certain areas, and that because of the effect on public opinion more money was made available.

There are many illustrations, and all the chapters have references. This book can be highly recommended to all those interested in a condition which certainly still exists in the Union of South Africa.

F.W.F.P.

UROLOGY BY MANY CONTRIBUTORS

Urology, Vols. 1, 2 *and* 3. Edited by Meredith Campbell, M.S., M.D., F.A.C.S. (Pp. 1-862, 863-1666, 1667-2356, with 1148 figures. South African price £25 10s. for set of 3 volumes.) Philadelphia and London: W. B. Saunders Company. 1954.

Contents: Volume 1. 1. Anatomy and Physiology. 2. Principles of Diagnosis. 3. The Pathology of Urinary Obstruction. 4. Embryology and Anomalies of the Urogenital Tract. 5. Infections and Inflammations of the Urinary Tract. 6. Infections and Inflammations of the Male Genital Tract. 7. Infertility in the Male. 8. Urinary Lithiasis and Foreign Bodies. *Volume* 2. 9. Injuries of the Urogenital Tract. 10. Tumors of the Urogenital Tract. 11. Neuromuscular Disease of the Urinary Tract. 12. Urology in the Female. 13. Urology in Infancy and Childhood. *Volume* 3. 14. Endocrinology in Urology. 15. Urologic Surgery. 16. Radiation Treatment of Tumors of the Genitourinary Organs. 17. Medical Diseases of the Kidney. 18. The Adrenals. Index to Volumes 1, 2 and 3.

The American publishers who induced Dr. Meredith Campbell to collaborate with 51 contributors in the production of yet another tome on urology are indeed to be congratulated. In this 'Urology' in 3 volumes we have an excellent and authoritative reference book which covers more adequately than ever before the entire scope of urology and its related basic and clinical sciences. Urology, like most divisions of medicine, has advanced and increased to such an extent that one author can no longer present all phases of the subject adequately. A one-volume, one-author attempt must of necessity be a 'Synopsis' or an 'Essential Urology' and much of the content is repetition from earlier works or information gleaned from others.

The 3 volumes comprise 18 broad sections, which are so comprehensive that each is subdivided into several chapters, each written by a different author. Few authors are responsible for more than one chapter.

To illustrate the scope and subdivisions of the sections one of

them will be outlined, viz: *Section X*, Tumours of the Urogenital Tract:

Chap. 1, Tumours of the Kidney, by Clyde L. Deming; Chap. 2, Tumours of the Ureter, by Winfield W. Scott; Chap. 3, Tumours of the Bladder, by Hugh J. Jewett; Chap. 4, Benign Hyperplasia of the Prostate Gland, by Robert V. Thomson and J. E. Ash; Chap. 5, Carcinoma of the Prostate, by William W. Scott; Chap. 6, Tumours of Seminal Vesicles, by Thomas E. Gibson; Chap. 7, Tumours of Penis, Urethra, Scrotum and Testis, by Archie L. Dean; Chap. 8, Tumours of Spermatic Cord, Epididymis and Testicular Tunics, by Thomas E. Gibson.

The editor in his preface claims that many of the contributions are classic monographs, and this is no overstatement of fact. Each chapter has been compiled by an authority in the particular topic. Except three, the authors are Americans, but they are of international stature. In fact this 'Urology' needs no better recommendation than the list of contributors.

A book of numerous contributions has inherent characteristics which some may deem disadvantages. There is some repetition and overlapping. Some chapters are better than others. The editor has succeeded admirably in selecting authors, getting the best from each, and arranging the text so that good continuity and sequence is maintained.

This 'Urology' covers the science in the wider sense and ample consideration has been given to allied and related subjects. The sections on Anatomy, Physiology, Neurology, Endocrinology and X-ray Therapy are well elaborated. A section of 12 chapters is devoted to the technique of urological operations, and operative procedures are outlined in several other chapters. The sections on Anaesthesia and Medical Diseases of the Kidney are desirable inclusions. Urological surgery probably more than any other branch of surgery requires careful consideration of water and electrolyte studies, and this has been dealt with in a thoroughly practical manner. There is in fact no modern trend either investigatory or therapeutically which has not received adequate attention. Each chapter is followed by a carefully selected list of references, which increase the value of the work as a reference book.

There is little in this work to criticize. The section on Parasitology, and in particular the chapter on Bilharziasis, is not as good as the rest. We recognize this because this disease is endemic in South Africa. The Editor might well have invited an authority from an endemic area to contribute the needed monograph.

The setting, type and editing make for easy reading. The illustrations are carefully selected and reproduced with great clarity. There is a full index of all 3 volumes at the end of each volume.

The book is dedicated to 'Those who may be helped by these volumes'. Their number is legion; anyone interested in this or any related subject can hardly afford to be without these volumes.

P.J.M.R.

THE DIENCEPHALON

Diencephalon: Autonomic and Extrapyramidal Functions. By Walter Rudolf Hess, M.D. (Pp. 79 + xii, with 33 illustrations. $4.00). New York: Grune and Stratton. 1954.

Contents: 1. Foreword. 2. Preface. 3. *Part* 1. Functions of the Diencephalon and Neighbouring Related Areas. 4. The Method of Localized Stimulation and of Destruction of the Stimulated Area in the Unrestrained Animal. 5. Synthesis of Experimental Results. 6. Autonomic Effects, Including Somatomotor Auxiliary Mechanisms. 7. Diencephalic Motor Innervation. 8. Autonomic Effects and Frontal Lobe. 9. Some Prospects for Future Research on the Branch. 10. *Part II.* Integration of Autonomic Functions. References. Index.

It is a pleasure to read this little book, which gives so much information on the diencephalon. The different methods used to clarify the function of the diencephalon are not only of interest to physiologists and pharmacologists but will become increasingly important to neurologists and psychiatrists.

The tremendous amount of work done by Hess and his co-workers can be realized fully only if one remembers how much time is spent on preparing stained sections of the brain and mapping out the different points stimulated. The exact localization of the points of stimulation is done by means of a series of photograms covered with coordinate nets. The cinematographic method of picturing all phases of the cats' behaviour after stimulation of the different parts of the diencephalon, is also described. These are only a few aspects of this extensive work.

The problem of direct comparison of anatomical substrata and physiological patterns has become more and more difficult since biochemical and physical methods have opened a new field of research technique.

The fundamental statement made by Hess that 'it can hardly be wrong to regard the diencephalon, rather than the cortex, as the true central organ of the body' becomes clear if one takes into consideration the numerous careful experiments and the author's concept of a 'centre'. The centre, according to Hess, 'unites the function of individual effector organs for the performance of a definitive task'.

It is to be hoped that in the future more books by W. R. Hess will be translated and edited in this clear and careful manner.

M.W.

BOOKS RECEIVED : BOEKE ONTVANG

Clinical Endocrinology. By Laurence Martin, M.D., F.R.C.P. and Martin Hynes, M.D., M.R.C.P. Eighth Edition. (Pp. 253 + viii with illustrations. 20s.) London: J. & A. Churchill, Ltd. 1954.

Pediatric Diagnosis. By Morris Green, M.D. and Julius B. Richmond, M.D. (Pp. 436 + xvii). Philadelphia and London: W. B. Saunders Company. 1954.

Kwashiorkor. By H. C. Trowell, *O.B.E.*, M.D., F.R.C.P., J. N. P. Davies, M.D. and R. F. A. Dean, Ph.D., M.R.C.P. (Pp. 308 + xii. 50s.) London: Edward Arnold (Publishers), Ltd. 1954.

Babies and Young Children. By Ronald S. Illingworth, M.D., F.R.C.P., D.P.H., D.C.H., F.R.P.S. and Cynthia M. Illingworth, M.B., B.S., M.R.C.P. (Pp. 360 + vii with illustrations. 18s.) London: J. & A. Churchill, Ltd. 1954.

Medicine in Its Human Setting. By A. E. Clark-Kennedy, M.D., F.R.C.P. (Pp. 276 with illustrations 13s. 6d.) London: Faber & Faber, Ltd. 1954.

The Diagnosis of the Acute Abdomen in Rhyme. 3rd Edition. By Zeta. (Pp. 96 + vii with illustrations. 7s. 6d.) London: H. K. Lewis & Co., Ltd. 1955.

The National Formulary 1955. The Pharmaceutical Society of Great Britain. (Pp. 210. 5s. Interleaved 8s.) London: The Pharmaceutical Press. 1955

CORRESPONDENCE : BRIEWERUBRIEK

MENINGO-ENCEPHALITIS DUE TO COXSAKIE B VIRUS IN SOUTHERN RHODESIA

To the Editor: With reference to the article on this subject by Drs. A. J. W. Wilkins *et al.*[1], several points arise of clinical interest, and of importance in that the diagnosis of Coxsakie B virus infection becomes clinically possible.

1. In these cases there is a marked incidence of isolated muscle spasm, pain and tenderness, of one or two days' duration, flitting from one muscle to another, involving several muscles during the course of the disease. Thus, one patient had painful spasm of the left adductor longus of 2 days' duration, followed in turn by involvement of the right pectoralis major, the right adductor longus, and the left sacrospinalis muscles. The affected muscles were tender. In acute anterior poliomyelitis, on the other hand, tenderness and pain are much more diffuse, and there is no flitting tendency. I feel this is an extremely important feature in the diagnosis of Coxsakie virus infection.

2. Vague abdominal pain of short duration, apparently due to involvement of retroperitoneal muscles, is often present. In

Bornholm disease, which is a Coxsakie virus-B infection affecting the diaphragmatic muscles, the thoraco-abdominal pain is severe and sharp.

3. Neck rigidity is transient and slight, and the posterior cervical muscles will be found to be spastic, painful, and tender. In like manner, stiffness of the back is not marked and is of short duration, thus differing from acute poliomyelitis.

4. Kernig's and Brudzinski's signs appear to be consistently negative in Coxsakie infection, in this respect also differing from poliomyelitis infection.

5. The duration of headache and fever, in my experience, may be up to 2 weeks. Cases are recorded of 4 weeks' duration.[2]

As Coxsakie virus infections appear to be becoming more widespread and prevalent, and as facilities for the rapid isolation of the organism and for the determination of neutralizing antibodies are not available, it is important that the clinical features of this group of illnesses should be widely appreciated.

Finally, may I make a plea against the almost universal practice of lumbar puncture in cases of doubtful poliomyelitis. It appears that this practice may result in converting a proportion of non-paralytic polio cases into paralytic cases. Thus, during a polio epidemic, it may be wiser to withhold this diagnostic procedure and rely upon clinical judgement.

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S. Rhodesia
20 January 1955

1. Wilkins, A. J. W. *et al.* (1955): S. Afr. Med. J., **29,** 25 (8 January).
2. Kenyon, H. *et al.* (1952): Lancet, **2,** 153.

TONSILS AND ADENOIDS

To the Editor: I am grateful to Dr. Philip Meyrick[1] for his courtesy in forwarding me a copy of a letter he addressed to you.

I specifically avoided the now commonly used expression Tonsilloadenoidectomy—a cumbersome word—because I dealt primarily with the tonsils. The word adenoidectomy could, with profit, have been omitted from the indications. The methods of adenoids vary with different surgeons.

Repeated attacks of Otitis media may have been classed as a positive indication but only after a serious attempt has been made to use conservative therapeutic measures, to determine the exact source of the discharge and to assess the state of the mastoid. I would never ask a colleague to remove adenoids without obtaining a definite answer to the above questions.

I deeply regret I consider myself unqualified to write on deafness, which, next to blindness, is a tragic affliction and should be considered, as Dr. Meyrick suggests, an urgent problem.

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28 January 1955

1. Meyrick, P. S. (1955): S. Afr. Med. J., **29,** 116 (29 January).

TWO CASES OF RUPTURED UTERUS

To the Editor: I should like to thank Professor D. Crichton[1] for his very logical letter in the *Journal* of 15 January. My intention was not to advocate an increase in the elective Caesarean section rate but to make a plea for a balanced outlook in cases such as the one in point. Professor Crichton has not put the emphasis where I intended it; namely, that having had a normal 6-lb. infant, the patient then had a still-born infant of 7½ lb. after a difficult forceps delivery with scarring of the cervix and possibly of the lower segment. I feel that here a case can be made out for an elective Caesarean section, especially as there was a reasonable chance that the third child would be still larger. In fact, it may be the more conservative line to take, especially as in hospital practice one may have to rely on the observations of less experienced individuals regarding the nature of the contractions.

Professor Crichton knows of the difficulty in differentiating the distal portion of the uterus and the upper vagina in the second stage. What may be thought to be a low sub-total hysterectomy may turn out to be a total, and *vice versa*. In my case, where closure of the vault was most important to avoid intestinal prolapse, only scar tissue and no cervical lip was felt on vaginal examination at the post-natal examination.

In conclusion, the contributing factors in a case such as the one reported point to the fact that an elective Caesarean section would be a reasonable method of delivery. Let me, however, reassure Professor Crichton that I believe that generalizations are dangerous, and that a patient must be guarded against unnecessary surgical assault.

James Miller

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1. Crichton, D. (1955): S. Afr. Med. J. **29,** 72.

TUBERCULOUS MENINGITIS

To the Editor: It was with grave concern that I read the article by Dr. Craig on Tuberculous Meningitis[1] which appeared in your issue of 15 January 1955. While admiring his resourcefulness in treating his cases under difficult conditions, it is disquieting that he should have instituted a method of therapy which, apart from being novel and convenient, has nothing to commend it.

It has been shown that it is extremely doubtful if any benefit can be claimed for the use of ACTH in the treatment of tuberculous meningitis.[2] That ACTH and cortisone can cause spread of pulmonary tuberculosis, while suppressing symptoms, is well known. Yet no mention is made in Dr. Craig's paper of chest X-rays in any of his patients.

It has also been demonstrated that intramuscular streptomycin and oral PAS do not penetrate the blood-brain barrier (hence the need for intrathecal streptomycin in the treatment of tuberculous meningitis). Therefore their inclusion in the regime of treatment (unless designed to combat hypothetical pulmonary tuberculosis) strikes one as being illogical.

INH has been shown to attain satisfactory concentrations in the cerebro-spinal fluid when administered orally. One is tempted to assume that Dr. Craig's patients survived because of INH, *despite* the rest of the scheme of treatment.

The average stay in hospital of the 8 cases was 62 days; yet the usual period of hospitalization in most centres is 9 to 15 months. To discharge patients from hospital with a severe attack of coronary thrombosis after 1 week's treatment would, I submit, be a not unfair analogy. In addition, the maximum follow-up period of the cases was only 10 months after discharge from hospital. In this hospital, cases are observed for a minimum period of 2 years after going home, as it is well known that any worth-while statistical appraisal of the results of therapy in tuberculous meningitis can only be based on long term observation because of the tendency to relapse.

Intrathecal streptomycin is still the cornerstone in the treatment of tuberculous meningitis to-day. As lumbar puncture in hospital practice (whether in Klerksdorp or Cape Town) is neither a time-consuming nor dangerous procedure, I fail to see why intrathecal theraphy was omitted from Dr. Craig's therapeutic scheme.

'It is amply apparent from the work of others that many patients with tuberculous meningitis may recover without any intrathecal treatment. Our object, however, must be to cure the highest possible number of patients and it has not been shown that this can be done without intrathecal treatment.'[3]

In South Africa tuberculous meningitis must surely be, after gastro-enteritis and the commoner severe infectious diseases, the greatest killer in children under 5 years of age, particularly amongst the non-European races. A survival rate of between 70% and 90% has been shown possible with the use of streptomycin (intrathecally and intramuscularly) together with oral PAS and INH, provided that diagnosis has been made early. At present it would be disastrous if we were to abandon a well-tried and effective scheme of treatment for one which is fraught with danger and has no virtue apart from its deceptive attractiveness.

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20 January 1955

1. Craig, C. J. T. (1955): S. Afr. Med. J., **29,** 62.
2. Lorber, J. (1953): Brit. Med. J., **2,** 1433.
3. Lorber, J. (1954): Lancet, **1,** 1151.